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WATER AND SALT TURNOVER IN CANE-CUTTERS WORKING ON THE COASTAL SUB-TROPICS OF AUSTRALIA.

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ALTHOUGH the survival of Europeans in Northern Australia is of some interest, there are few data on the physiological adjustments of those living in the region. Early work by Sundstroem (1926) at Townsville related mainly to biochemical estimations on blood rather than to physiological patterns. Lee (1940) has estimated human sweat loss in laboratory situations, but the subjects were under no incentive to sustained hard work. In 1949 McPherson published an account of field studies of fitness for work, blood pressure and heart rates in the tropics; but water and salt turnover was not measured.

The estimations reported here were made to ascertain the likely maximum intake of water by adult males in a hot climate. This datum is relevant to maximum fluoride intake and to the possibility of fluorosis, should fluoridization of water supplies be undertaken to reduce dental caries. The water, salt and nitrogen turnover was estimated on 10 men performing hard work in a hot and humid environment near the coastal town of Bundaberg (25° south latitude). The observations probably indicate the maximum sustained work performed in the sub-tropics,

and this is supported by comparisons made with mill and factory workers and school children. In the far north the water demand may be greater, though the heat of the day is often avoided there by working early and late rather than in the midday sun.

Methods.

The fluid turnover of 10 acclimatized men working in summer sun temperatures up to 92° F. (dry bulb) and 82° F. (wet bulb) in a relative humidity of 64% was measured at the end of the cane-cutting season at Bundaberg. Changes in weight (in shorts) between 7 a.m. and 4.30 p.m. were estimated, a balance sensitive to 50 grammes being used. Urine output, fluid intake and urine constituents were also determined. Urine was collected in graduated bottles, fluid was drunk from graduated containers and recorded on a tabulation sheet, and food (lunch only) was weighed. The urine volume, measured intakes of fluid and losses of weight served as internal checks on each other. Since the subjects lived in many scattered places, it was not possible to estimate the breakfast and dinner intakes of food. The fluids taken during twenty-four hours were, however, recorded. Such field studies as this have many sources of inaccuracy; but the subjects were keen and cooperative, and the internal checks of the method indicate that the accuracy approached 10%.

The men worked beside or amongst standing cane where wind convection was low. Lines for trams had to be moved, cane was cut, and the bundles were lifted and stacked upon the trucks. Measurements were made during

TABLE I.
Fluid Turnover During Forty Man-Days of Cane Cutting at Bundaberg in Summer.¹

Maximum Temperature.	Day.	Weight Loss. 7 a.m. to 4.30 p.m. (Kilograms.)	Urine. 7 a.m. to 4.30 p.m. (Litres.)	Weight Loss minus Urine. ² 7 a.m. to 4.30 p.m. (Kilograms.)	Total Fluid Intake. 7 a.m. to 4.30 p.m. (Litres.)	Apparent Sweat plus Carbon Dioxide Loss. 7 a.m. to 4.30 p.m. (Litres.)	Twenty-four Hour Fluid Intake. (Litres.)	Twenty-four Hour Urine Excretion. (Litres.)
88° F.	First.	2.07 ± 1.01	0.424 ± 0.113	1.63 ± 1.10	6.15 ± 1.01	7.78 ± 1.57	7.96 ± 0.95	1.14 ± 0.26
91° F.	Second.	2.93 ± 1.36	0.590 ± 0.092	2.32 ± 1.37	7.33 ± 1.15	9.65 ± 1.56	9.65 ± 1.56	1.29 ± 0.17
90° F.	Third.	2.29 ± 0.37	0.639 ± 0.161	1.65 ± 0.43	6.24 ± 1.06	7.87 ± 1.16	8.23 ± 1.50	1.33 ± 0.25
92° F.	Fourth.	3.32 ± 0.91	0.513 ± 0.103	2.80 ± 0.96	8.30 ± 1.11	11.10 ± 1.44	(10.80) ³	—
Mean	2.65	0.541	2.10	7.01	9.10	8.61 (9.16) ³	1.25

¹ Means and standard deviations are tabulated for each day.

² Corrected for specific gravity.

³ Estimate (since twenty-four-hour sample not obtainable on Saturday).

four working days at the beginning of December. There was some cloud and a cool south wind during the first three days, but the sun was not obscured and there was little wind on the fourth day. The noonday humidity reached 50% to 60%, with air temperatures, taken in the field by sling psychrometer, of 88° to 92° F.

Results.

The average age of the gang was 33.4 years (twenty-seven to forty years), and the average weight (stripped) and standard deviation were 71.51 ± 7.64 kilograms. The men were of English, Irish, Scottish or German stock. During the week of these studies the average cut per day by each man was 9.4 ± 1.1 tons of cane. Oral temperatures were below 99° F. during work.

Fluid intake for twenty-four hours (apart from water derived from food and metabolic water) averaged 8.6 litres for three relatively cool days and an estimated 9.16 litres over four days (Table I).

At work 83% of the fluid was taken as water; but at home water comprised only 37% of the intake (Table II). Tea made up 17% of the fluids ingested at work and 28% of the fluids taken at home. In this type of occupation, with high rates of sweating, an average of only 19% of the total fluid ingested was tea.

TABLE II.

Amounts of Water, Tea and Other Fluids Taken by Ten Cane Cutters During Work Days in Summer.

Fluid.	At Work: 7 a.m. to 4.30 p.m. (Litres.)	At Home: 4.30 p.m. to 7 a.m. (Litres.)	Total for Twenty-four Hours. (Litres.)
Water	5.73 ± 1.32	0.83 ± 0.57	6.56 ± 1.38
Tea	1.16 ± 0.78	0.61 ± 0.57	1.77 ± 0.67
Milk, soft and hard drinks	0.06 ± 0.05	0.77 ± 0.44	0.83 ± 0.48
Total intake (mean)	6.95	2.21	9.16

At the end of eight hours' work an average of 2.11 kilograms' weight was lost in excess of the food and fluid replacement (Table I), after allowance had been made for urine production. The total loss (weight loss plus fluid intake) requires some correction for metabolic and food water (Morrison, 1953). Since the subjects were working in the field and full metabolic studies were not possible, some assumptions are necessary.

The work performed is likely to have reached 400 kilocalories per hour, or 3200 kilocalories in the eight-hour working day. During work the respiratory quotient approximates 1.0. The carbon dioxide breathed out would then equal the volume of oxygen taken in; but each mole of carbon dioxide expired would remove 12 grammes of

carbon. For 3200 kilocalories used, 0.33 kilogram of carbon would be expired.

Metabolic water is evaporated either in sweat or through the respiratory tract, and is included in the weight changes. No faeces were passed during the work period. The estimated mean loss of water by evaporation during eight working hours may be calculated as follows:

	Kilograms.
Mean weight loss (corrected for food intake)	2.65
Less urine excreted	0.54
Plus mean fluid intake	2.11
Mean weight loss (corrected for food intake and urine excretion)	7.01
Less carbon lost in respiration	9.12
Mean loss of water by evaporation	0.33
Mean loss of water by evaporation	8.79

The estimated mean evaporative loss of water (8.79 kilograms) was considerably exceeded by some of the subjects. One man weighing 78 kilograms drank 11.7 litres, so that he probably sweated more than 12 litres in twenty-four hours.

On the average, nitrogen equivalent to 120.6 grammes per day was excreted, and 254 milliequivalents of sodium were passed (Table III). The ratio of sodium to potassium in the urine ranged from 1.5 to 3.0 over the twenty-four hours, but in the work period the ratio was lower than in the rest period at night.

A survey of occupations in which men worked in the heat was made in Bundaberg for comparison with cane cutting. In the sugar mills, men working beside furnaces, dryers or evaporating vats were exposed to temperatures of 90° to 92° F. (dry-bulb) and 82° to 85° F. (wet-bulb) during the afternoon. During the work-shift, water intake was estimated (from water bag fillings) to vary from two to four litres. The maximum fluid intake at a drying furnace was six litres per twenty-four hour day.

Another comparison was made in the Bundaberg summer environment, the subjects being 26 school children aged eleven to thirteen years. The children were selected for their intelligence. Graduated beakers were given to them, and they recorded their fluid intakes over two periods of twenty-four hours. More than one litre of water and about 250 millilitres of tea were taken by these children daily (Table IV).

Discussion.

The ten cane-cutters studied were incentive-paid workers, who had become acclimatized during six months of cutting, and they had all cut cane during several seasons. Each day they produced over one litre of sweat per hour, and they returned home on the average 2.1 kilograms lighter than they were at the beginning of the day. Pitts, Johnson and Consolazio (1944) have observed that men replace

TABLE III.
Nitrogen, Sodium, Chloride and Potassium Excretion.¹

Urine Volume. (Litres per 24 Hours.)	Nitrogen ; Protein Equivalent. (Grammes per 24 Hours.)	Cl ⁻ . (Milliequivalents per Litre.)	Na ⁺ . (Milliequivalents per Litre.)	K ⁺ . (Milliequivalents per Litre.)	Sodium- Potassium Ratio.	K ⁺ . (Grammes per 24 Hours.)
1.386 ± 0.278 (1.000 to 1.740)	120.6 ± 34 (84 to 200)	171 ± 26 (113 to 199)	183 ± 24 (162 to 226)	80.5 ± 11 (61 to 95)	2.28 (1.5 to 3.0)	4.4 ± 0.94 (3.1 to 5.7)

¹ The mean, standard deviation and range of urine constituents are tabulated for sixteen man-days.

only about two-thirds of the water lost during work. The cane-cutters took a large dinner at night and were back to standard weight in the morning. If the water sweated (8.79 kilograms) were all vaporized, about 5200 kilocalories could be lost in twenty-four hours. Since radiant heat gain (at about 600 kilocalories per square metre per hour) and convection loss were also present, the sweat output was probably used for cooling at 90% efficiency, if it is assumed that 2400 kilocalories of metabolic heat and over 2000 kilocalories of radiant heat were dissipated in eight hours and 800 kilocalories of external work done. The men wore singlets and shirts which prevented sweat from dripping away, so that evaporative cooling resulted from most of the sweat secreted.

TABLE IV.
Various Types of Fluid Taken During Summer by School Children
Aged Eleven to Thirteen Years (Means and Standard Deviations).
Air Temperature Maximum, 91° F.

Fluid.	December 1. (Millilitres.)	December 2. (Millilitres.)
Water	1023 ± 468	1113 ± 483
Milk	303 ± 244	261 ± 215
Soft drinks ..	203 ± 216	142 ± 160
Tea	246 ± 234	288 ± 243
Total	1775 ± 460	1804 ± 430

The total fluid turnover ranged in individuals as high as 11.7 litres per twenty-four hours. On the hottest day (which was not severely hot at 92° F. dry-bulb) the mean evaporative loss during the work period was 10.9 kilograms per 70 kilograms per eight hours. The thirst mechanism does not keep pace with this rate of sweat secretion; but the urine volume for twenty-four hours (1.25 litres) indicates that mild water diuresis was achieved after work (night urine, 710 millilitres; day urine, 541 millilitres). Some excess intake occurred, therefore, after 4.30 p.m.; but the members of the gang studied did not take beer until the week-end, since they found that drinking alcohol during the week reduced the tonnage of cane that could be cut.

From the point of view of fluorine intoxication, the ingestion of tea could be important. The men studied at Bundaberg took an average of 1.77 ± 0.67 litres per day of tea. One subject drank 3.92 litres of tea in twenty-four hours. The amount of fluorine in tea depends upon the brand and the strength of infusion, and ranges from 0.5 to 2.2 milligrammes per litre (Macfarlane and Harvey, 1957). Ingestion of 1.1 to 3.9 litres of tea could represent intakes of 0.5 to 8.7 milligrammes of fluorine each day. Such quantities would be obtained from one to 17 litres of water containing 0.5 part per million of fluorine. Tea could therefore contribute about half the fluorine intake of heavy tea drinkers if water was fluoridized. Galagan (1953) has investigated the fluorosis occurring in children who were exposed to water containing 0.4 to 1.2 parts per million of fluorine in hot regions of Arizona. He found that water containing up to 0.6 part per million of fluorine produced negligible mottling in children aged up to sixteen years. Children in Australia drink some tea, so that their fluorine intake could be rather greater than that of children in Arizona, who probably drink little tea. In the adult,

fluorine is stored in bones and does not increase mottling of teeth.

These estimates of water and tea intake would give a better indication of the fluorine load likely in adults than fluorosis studies in children can give.

There was no deficit in sodium or chloride excretion amongst these men. No conscious effort to add salt to the diet was made by the cutters; but the large quantities of food taken were evidently salty enough to provide a mean urinary excess of about 13.8 grammes of sodium chloride per day. A sweat loss of 8.78 litres per day would, at 0.1% sodium chloride concentration, require a further 8.78 grammes per day intake. They were acclimatized for five or six months, so that their salt excretion would be minimal (Weiner and van Heyningen, 1952). Several men reported that if they added more salt to the diet, they suffered swollen feet. This seems likely to be the consequence of the long-persistent action of aldosterone. Sodium retention may last up to forty-eight hours after heat and sweat have brought about a reduction of the sodium-potassium ratio in the urine (Macfarlane, 1956). Salt retention allows water retention and leads to oedema. The output of 4.4 grammes per twenty-four hours of potassium is a little above the normal range, and is probably a reflection of the food intake, rather than a sign of cellular potassium mobilization.

In the studies made by Adolph (1938) on water turnover in men marching at about four miles per hour in the desert, the hourly sweat loss was estimated at 1.02 kilograms per hour over a temperature range from 83° to 108° F. In later studies, Adolph (1947) reports mean rates of 0.87 kilogram per hour at higher temperatures (93° to 114° F.) when men walked in the sun, but less enthusiastically than in the earlier studies. This led Adolph to conclude that sweating increased at the rate of 22 grammes per 70 kilograms per hour per degree Fahrenheit in men walking in the desert. This equals 0.92 kilogram per 70 kilograms per hour at 100° F. The Bundaberg workers evaporated water during work at a mean rate of 1.06 kilograms per 70 kilograms per hour at temperatures up to 92° F. (dry-bulb), 82° F. (wet-bulb). The work performed was apparently harder than that of Adolph's subjects, and the effect of humidity may have increased the heat stress at Bundaberg.

Cane-cutters probably perform the hardest work undertaken on the coastal belt of Australia, and the metabolic parameters determined approach the maximum for work in this type of environment. The men had apparently reached a steady state with a turnover of water about five times that of sedentary workers in the same environment.

Summary.

1. Ten cane-cutters were found, during summer work at 25° south latitude, to have a mean intake of 7.01 ± 1.08 litres of fluid during eight hours' work. They lost 2.11 kilograms of weight in excess of replacements during the work period. The estimated mean evaporative loss of water for eight hours' work was 8.79 kilograms.

2. The maximum fluid intake for twenty-four hours was 11.7 litres, and the mean for three moderately hot days 8.61 ± 1.3 litres.

3. An average of 1.77 ± 0.67 litres of tea were taken daily. The maximum tea intake was 3.92 litres. Tea represented 19% of the fluid ingested.

4. The protein equivalent of the urinary nitrogen excretion was 120.6 ± 34 grammes per twenty-four hours. Amongst electrolytes, 4.4 ± 0.94 grammes of potassium, 5.8 ± 0.44 grammes of sodium and 8.4 ± 0.91 grammes of chloride were excreted in twenty-four hours. The sodium-potassium ratio in the urine averaged 2.28. The urine volume averaged 1.25 litres per twenty-four hours.

5. The relation of these rates of fluid ingestion to fluorine intake is discussed.

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DR. BELLAMY OF PAPUA: II.¹

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Public Health.

BEFORE the work of R. L. Bellamy in the sphere of public health is described, it is necessary briefly to indicate the attitude to this field of endeavour in early British New Guinea.

Sir William MacGregor, as well as being the first Administrator from 1888 to 1898, also acted as the Government Medical Officer. His interests were mainly exploration and administration with the object of pacification, but he occasionally noted the disease conditions in the various places he visited. In the earlier annual reports his notes on health were concerned in the main with Europeans, and the annual medical and sanitary expenditure for the Possession was £100, increasing to £200, in a total budget of £15,000. MacGregor pointed out that the sum of £100 might not always suffice for the medical and sanitary service. This small vote could continue only so long as the Administrator happened to be a medical man, willing to do the medical work without remuneration. The Government would probably always require at least one medical man, as had been considered necessary even with the small establishment of Sir Peter Scratchley. MacGregor also stated that £500 was probably the minimum value of the services of a medical officer.

Late in 1894 the Government of British New Guinea was informed by a government official of German New Guinea that smallpox had been present in the latter territory for a year or more and that it had reached a point on the coast within 100 miles of British New Guinea. This caused much anxiety and

fear that the disease would enter the Possession. However, it was decided not to survey the border area, as none of the native government servants had been vaccinated. The disease did not reach British New Guinea, and as no further information was received from German New Guinea, it was presumed to have disappeared from that territory in 1895. As soon as vaccine lymph was obtained the constabulary and other government servants and prisoners were vaccinated. Some of the older natives of the British Possession remembered smallpox from an epidemic which had occurred about 1870.

In MacGregor's early opinion the climate of New Guinea was healthy apart from the fever. At the end of his ten years in the country he described the various diseases he had seen and maintained that fever was the most frequently encountered disease.

One of MacGregor's resident magistrates held medical qualifications, and this officer, Dr. J. A. Blayney, became Chief Medical Officer, as well as remaining the Resident Magistrate of the Central Division, when MacGregor left the Possession. The medical and sanitary vote remained at £200, of which nearly one-half was unexpended in the year 1898-1899. Mr. C. Vaughan was appointed as the Medical Officer at Samarai in December, 1899. Blayney's first report, in 1901, described the prevalence of various diseases amongst the native people, and deplored the absence of hospitals for Europeans at Port Moresby and especially at Samarai, which had the larger population. In this year the vote for the Medical Department rose to £750, of which £130 was not expended. In the following year (1900-1901) Blayney went on leave and Dr. A. J. Craigen became Acting Chief Medical Officer. Craigen reported that Blayney had accompanied the Goaribari expedition and had successfully administered daily doses of quinine as a prophylactic for malaria; his professional services were required only for an arrow wound of a trivial nature, received by one of the policemen.

Blayney resigned at the end of his leave, and Craigen remained as the Acting Chief Medical Officer. The vote for the Medical Department increased to £1887 10s. of a total of £31,551 5s. The Acting Administrator, Winter, in reviewing the report of the Chief Medical Officer, noted that "even at Port Moresby, many natives will not go to the Medical Officer and ask for his help". Further, Winter stated that "the observation of the Medical Officers of necessity does not extend very far. Throughout the greater portion of the Possession the native population is not under medical supervision".

In the year 1902-1903 Craigen was appointed Chief Medical officer and Dr. J. T. Hancock became Government Medical Officer of the Eastern Division, stationed at Samarai. The European hospital at Samarai had been built by public subscription, but the one proposed for Port Moresby had not been commenced owing to the lack of carpenters. A temporary hospital had been necessary at Port Moresby in 1897 to deal with a sudden influx of sick miners who had made their way inland from the south coast. This hospital consisted of the local gaol, the warder's cottage, and two tents. In 1902-1903 sanitary boards were created at Port Moresby and Samarai and a pan system of disposal was introduced. In the following year a native hospital at Samarai proved a great success, and it was noted by Barton, who became Administrator in 1904, that both European and native hospitals were about to be commenced at Port Moresby. In the year 1904-1905 Dr. C. C. Simson was Chief Medical Officer, and Dr. R. F. Jones Medical Officer at Samarai. In the following year Dr. N. C. Beaumont became Chief Medical Officer and Jones remained at Samarai.

This is by no means a complete review of the history of medical activity in British New Guinea, but it serves to show the early attitude of the few medical men in the country—and their sojourns were brief. Hospitals are commonly regarded as the first signs of the stirring of public health in a community. The Administration had been present in the Possession for about fourteen years before a European hospital was built at Samarai, and that by public subscription. Hospitals for native patients followed. A similar length of time had elapsed before an organized system of sanitation was introduced to the main centres of European population. There was no medical patrolling such as developed later, and the recording of disease incidence was qualitative. Populations were estimated and there was no census. There was no record of native births and deaths apart from those occurring in gaol or in government employment. The one major public health achievement had been the freeing of Samarai from endemic malaria by draining and filling in the swamp on that island. This work was commenced on the

¹ A full bibliography has been lodged in the Mitchell Library, Sydney, and in the library of the School of Public Health and Tropical Medicine, Sydney.

recommendation of MacGregor before the mosquito transmission of malaria had been discovered.

Thus it was at a time when there were the first stirrings of a public health consciousness that Bellamy, in 1905, was offered the position of officer in charge of a newly-built special hospital in the Trobriand Islands. It must be remembered that at this time Bellamy had practically completed his medical training at Edinburgh, but had not qualified. It was five years since he had left Edinburgh. His work on the control of venereal disease in this island group will be described as the first of his public health activities.

Venereal Disease.

In the early years of this century the Trobriand Islands were visited periodically by the Resident Magistrate of the South-eastern Division of British New Guinea, who had his headquarters at Woodlark Island. In 1905, The Honourable M. H. Moreton, a younger brother of the Earl of Ducie, who held this office, reported that the population of these islands was decreasing. He stated that syphilis, of which he thought there was a great deal, might be blamed for some of the deaths, and also to a small extent, for a decrease in the birth rate. Moreton also reported that Manilamen and Malays, engaged in collecting chalk fish, were a great source for the spread of contagious disease amongst the natives. He understood that a doctor was to be sent to the Trobriands to combat the syphilitic diseases, but he pointed out that, if the coloured native traders were not put under supervision, the spread of the disease would be encouraged with one hand whilst an attempt was made to stop it with the other. Also in 1905, the Chief Medical Officer reported that venereal disease was very prevalent in the Trobriand Islands (Simson, 1905).

The Administrator of British New Guinea, Captain F. R. Barton, visited the Trobriand Islands with Moreton and chose a site for a proposed special hospital to eradicate venereal diseases. He was convinced that these diseases were prevalent in the Group (Barton, 1905a). Barton pointed out that the control of venereal disease had not only a humanitarian aspect, but also was necessary for the future prosperity of the country. He hoped that the funds at the Government's disposal would be sufficient to stamp out these "baneful diseases". Moreton was instructed to build this special hospital at Losuia on Kiriwina, the main island of the Trobriand Group.

A similar hospital was proposed for Samarai. Here Dr. R. F. Jones had come to replace the previous Government Medical Officer, Dr. Hancock, who had died from malaria. Dr. Jones would be in charge of the special hospital at Samarai. He had wide practical knowledge of tropical diseases (Barton, 1905a). A "Special Hospitals Fund" was created after permission had been obtained to set aside £4600 out of the surplus balance for the Territory for the year 1903-1904. This fund provided the money for erection of the two hospitals, for equipment, and the salary of an additional medical officer. During the first year of operation of these hospitals £1042 was expended (Barton, 1907; British New Guinea, Report for 1905-1906).

On August 17, 1905, Barton wrote to Bellamy, who was then engaged in supervising the work on the Yodda Valley Road prior to leaving for England to complete his medical training. He informed Bellamy that permission had been obtained to start two lock hospitals—one at Samarai and the other in the Trobriands. Barton offered him the appointment of officer in charge of the latter hospital with a salary of £350 a year, with Bellamy retaining his appointment as an Assistant Resident Magistrate. The appointment would be for only one year for certain, but would probably be for three. The Administrator stressed that tact would be necessary in handling the Trobriand Islanders, and that the object would be to make the hospital as little unpopular as possible. He thought that the people would probably come forward voluntarily for treatment provided that a tactful approach was employed. Barton said he would be relieved if Bellamy took the billet, as he knew how well Bellamy always got on with the native people. Enclosed with this letter was a memorandum on the venereal diseases as seen by Dr. Jones at Samarai. Bellamy was requested to be at Samarai by September 30, 1905, if he accepted the appointment. He was informed that the hospital buildings and residence would probably be completed by the time he arrived in the Trobriands. Barton added that the Wesleyan Mission was entirely sympathetic with the projected special hospital, and that Bellamy would be sure to find the Reverend M. K. Gilmour helpful. Bellamy accepted this offer and sent his reply by the suggested special messenger overland to Port Moresby.

Meanwhile, Moreton loaded up the ketch *Siai* at Samarai with timber, iron etc., and at Hughes Bay on Fergusson Island, filled the ship with sago thatching for the hospital wards and the doctor's residence. He arrived at the Trobriands on August 22, and found that the Reverend M. K. Gilmour had commenced building the hospital and was so far advanced that thatching could be begun straight away. When Bellamy arrived on the steamship *President* on October 6, the dispensary, which was to be constructed with European materials, had not been built, but this was erected by Gilmour with the help of his mission carpentry class (Moreton, 1907; Anonymous, 1907).

The completed hospital consisted of buildings, mostly made from native materials, as follows: doctor's house and kitchen; three separate houses as wards for male patients; one building for female patients away from the other wards with a coral wall around it; two warders' houses; dispensary and store made from sawn timber with an iron roof. There was also a coral jetty 55 yards long, built by the crew of the *Siai* and prison labour. This jetty provided a landing stage at the northern end of the Trobriand lagoon.

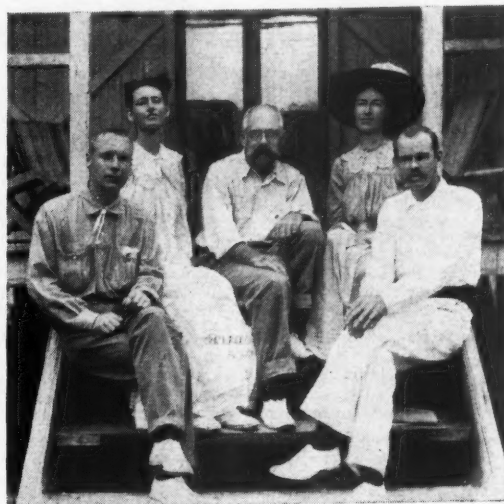


FIGURE VII.

Early Trobriand Island identities on the steps of the Mission house in 1905. Top left, Mrs. Gilmour; centre, the Honourable M. H. Moreton; top right, Miss Corfield; bottom left, R. L. Bellamy; bottom right, the Reverend M. K. Gilmour.

Bellamy wasted no time in settling in, and in the week after his arrival he accompanied Moreton and two or three native police to the village of Sinaketa, using a whaleboat which Bellamy had brought with him. Some opposition was met at Sinaketa, where the chief said that there was no one sick in the village. However, the chief was placed in the charge of the native police while Moreton went around the village with a boy. By the end of the day they had managed to bring back six girls and ten boys to the hospital. Moreton pointed out that they could not use too much coercion. By October 26, there were 26 female and 27 male patients in the hospital (Moreton, 1907).

When Moreton left, Bellamy found himself in charge of a special hospital with the task of eradicating venereal disease from a group of about 10,000 native people. Although Barton had said in his letter that Bellamy would retain the appointment of Assistant Resident Magistrate, this information apparently did not reach the Government Secretary's Department, and Bellamy was not reappointed to this office until some time later. He had no detachment of armed native constabulary for the first few years and had to rely on the village constables. Although Moreton reported that the village constables in the Trobriands were becoming of some use in 1905, the chief who had given some opposition at Sinaketa was also a village constable. The Europeans on the Trobriands at the time of Bellamy's arrival numbered 12, of whom three women and one man were at the mission station. The remaining eight males were engaged in trading and pearling.

The legal enactment upon which the attack on venereal disease was based was a regulation made by the Native Regulation Board (Number 1 of 1904, *British New Guinea Gazette* of September 10, 1904), which dealt with venereal diseases. Barton (1907) summarized some of the provisions of this regulation as follows:

Natives who are suffering from venereal disease are bidden to make the fact known to the nearest Village Constable, whose duty it then becomes to take the sick person to the nearest European Magistrate. It remains with the latter officer to decide whether the person is to be examined by a doctor, and if he should so decide, and if the doctor should subsequently certify that the person is afflicted with V.D., the Magistrate is empowered to issue an order that the native should be confined and kept in custody for treatment until cured or discharged.

In his reports Bellamy (1908, 1910) told of the difficulties which he encountered with native people coming into contact for the first time with a medical man of the Government. There was an uphill fight for half a year against native prejudice and superstition. Many of the first patients, brought in under pressure, had venereal disease of long standing. Patients were willing to give white man's treatment a trial for three days; then their gardens and their fishing called and they slipped away in the night, only to be brought back next day.

The hospital was up against some very powerful professional reputations—up against men who could remove cooking pots the size of hip baths from a man's inside; up against a man who could breathe three times on a leg, and remove a stone weighing several pounds.

In addition, the measure of a Trobriand man's strength was his garden, and if time had to be spent in hospital his garden suffered.

Then, one by one, the cured patients began to return to the villages and popular prejudice began to weaken. Bellamy soon found that to be a success the officer in charge of the hospital must be able to speak to the people direct and not through an interpreter. In learning the language Bellamy probably received help from the Reverend M. K. Gilmour, and the Reverend Fellows had published a grammar and vocabulary of the Kiriwina dialect in 1902, which was more detailed than the one prepared by MacGregor in 1892.

When Barton visited the Trobriands in December, 1905, he found that Bellamy had succeeded in gaining the confidence of the inhabitants beyond his most sanguine expectations, and that (only two months after the hospital had opened) patients were beginning to come voluntarily for treatment.

At the time when Bellamy began his work on the control of venereal disease in the Trobriand Islands, there were two qualified medical men in the Medical Department of British New Guinea—Jones and Beaumont. Jones (1907) was impressed with the difficulty of diagnosing the venereal diseases seen at Samarai. Rarely were cases of a simple character seen, and every possible complication was found. He had not seen any syphilis, or any of the well-known after-effects of this disease. He found that ulcerating granuloma of the pudenda was quite common. His cases consisted of gonorrhoea, chancroid and other venereal diseases. Both Jones and Beaumont, who was the Chief Medical Officer, doubted the effect of treatment in reducing the amount of venereal disease in the absence of measures to prevent the introduction of these diseases into the country and their spread by persons who knew they were infected. Barton (1907) stated that this question had been fully considered before the hospitals were started, and the required measures for meeting it were framed, but these had not been approved. He was probably referring to the "Undesirable Aliens Act for the Protection of the Trobriand natives", upon which subject Beaumont submitted a report to the Royal Commission for Papua in 1907.

Bellamy found that the Trobrianders had no knowledge of how venereal disease was spread. When he instructed his patients on the method of its transmission they were incredulous. He also taught the village constables their duty regarding the reporting of cases of venereal disease. Barton (1907) noted that in the first nine months of the hospital's existence 349 venereal disease patients had been admitted and 330 discharged. In Bellamy's report for 1906-1907 he commented on the difficulty of classification, but listed chancroids, syphilis, ulcerating granuloma and "other venereal diseases". He stated that gonorrhoea had, at the moment, nearly disappeared. By the middle of 1907 there was no more running away from hospital; but there was still some difficulty in persuading the women to come forward voluntarily for treatment. The patients were kept in the hospital until they were cured or died.

In October, 1906, the Royal Commission on Papua took evidence at Losuia, and Bellamy stated that about 10% of the

whole population of the Group, men, women and children, suffered from venereal disease. He said that the oldest Trobriand men with whom he had spoken about venereal disease knew about it when they were boys. It would not be fair to blame the present generation of white men for introducing it. Bellamy thought that the disease was probably brought in in the past by whalers calling at the Laughlan Islands. It would spread from the Laughlans to Woodlark, from Woodlark to Kitava and then to Kiriwina. Bellamy thought that syphilis was decreasing (Mackay *et alii*, 1907). Commissioner Mackay (1909) later commented on Bellamy's optimism about the results of the work on the control of venereal disease:

Mr. Bellamy, young and enthusiastic, thinks it can be stamped out. Dr. Jones, viewing it from the standpoint of a wider experience, hopes at best to hold it in check.

Bellamy considered every native to be a suspect subject to the Native Regulation Board's Regulation Number 1 of 1904 concerning venereal disease. On January 1, 1908, he set out to examine every man, woman and child in the 156 villages and hamlets in the Trobriand Islands. In the first six months of the year he examined 4976 persons. He entered their names in a register, which he stated was practically a census. He admitted 261 persons with venereal disease to hospital in the year 1907-1908, and of these cases 42 were readmissions as fresh infections or relapses from a group of 650 former patients. To cope with the nursing of these patients he had the assistance of one woman and three men—Trobriand Islanders, whom he had instructed in the necessary nursing procedures. In 1912 he had one male and one female orderly on the hospital staff. They had been assisting him since the hospital was opened seven years before and had become expert dressers. Bellamy said that if the hospital was closed, these two would probably set up in practice on their own and treat venereal diseases in return for payment in *kaloma* belts and tomahawk stones.

During 1909 Bellamy went on six months' leave—his first leave in over four years of service. He was relieved by Mr. J. C. Watt, a patrol officer, who had previously been a clerk in the Treasury at Port Moresby. As Watt arrived on the *Merrie England* on April 3, and Bellamy left on the next day, there can have been little personal instruction in the duties Watt was to perform.

On his return from leave Bellamy continued his systematic examination of the Trobriand Islanders, and the results were tabulated in his reports. Table I presented here shows the results

TABLE I.
Incidence of Venereal Disease in the Trobriand Islands, 1908 to 1915.

Year Ended June-30.	Number Examined.	Cases of Venereal Disease.	Percentage of Cases of Venereal Disease.	Number of Non-Venereal Genital Sores.
1908	5057	261	5.22	43
1909	5329	130	2.45	28
1910	6337	152	2.57	54
1911	6301	98	1.55	—
1912	7079	115	1.62	(† 28)
1913	6545	123	1.87	35
1914	6349	97	1.41	14
1915	7414	102	1.37	40

† Includes ten cases among labourers from outside the Group.

of his examinations over the years from 1908 to 1915. There was some difficulty at first, as the natives attempted to avoid examination; but after a few had been punished, Bellamy was able to obtain 100% attendances at his visits of inspection.

The venereal diseases diagnosed by Bellamy and listed in his annual reports were as follows: chancroids, gonorrhoea, syphilis, venereal wart and ulcerating granuloma. He made several comments in his reports on the clinical features of some of the venereal diseases as seen in the Trobriand Islands. His remarks on the difficulty of classification were in accord with those of Dr. Jones at Samarai; but as time went on he was able to eliminate the classification of "other venereal diseases" which, in the earlier years, appeared in his lists of diseases diagnosed. On following up his patients discharged from hospital as cured, he considered that a figure of 10% or less requiring readmission for relapses or fresh infections was satisfactory. He stated that ulcerating granuloma of the pudenda was incurable unless it was treated in the early stages. Bellamy

also commented upon the relative rarity of purulent urethral infections in both male and female natives. He said that he felt sure that there was an immunity which was, in all probability, an acquired one. He defined "chancreoid" as "an ulcerative lesion of the genitals, contagious and destructive of tissue, strictly localized and without any of the constitutional effects which distinguish the hard or syphilitic chancre".

With regard to Bellamy's diagnoses, the one disease which obviously requires comment is syphilis, although it comprised only a small proportion of the cases he reported. It is the present-day opinion that there is very little or no syphilis amongst the native peoples of Papua. At the time when Bellamy commenced his work in the Trobriands, Dr. Jones had not seen syphilis at Samarai, but reported the occurrence of this disease shortly afterwards. The Chief Medical Officer, Simson, had reported a case of syphilis in a native prisoner in 1905. Sir William MacGregor, after three months in Papua, had stated in 1889 that there was little or no venereal disease amongst the natives. In subsequent reports on the Trobriand Islands, which he later visited on several occasions, MacGregor made no mention of venereal disease. Rather, he remarked on the decidedly healthy population of Kiriwina, but he did not report making any examination for venereal disease. It appears that the first diagnosis of syphilis was made in the Trobriand Islands by Moreton, the Resident Magistrate of the South-eastern Division. Dr. Jones (1911) reported that not more than 2% of the patients of the special hospital at Samarai suffered from syphilis. He also stated that Bellamy had seen people with hereditary syphilis in the Trobriands.

Dr. A. Breinl, who was the first director of the Australian Institute of Tropical Medicine, in 1912 examined with Bellamy the patients in the special hospital at Losuia and in other parts of the Trobriand Islands. His report (1913) stated that he had found three cases of undoubted syphilis in three different villages in Papua, but did not give their location. In a later report Breinl (1915) noted that many of the manifestations of the late stage of yaws were very similar to syphilis, which was difficult to diagnose in a native community where various ulcers closely resembled syphilitic lesions. Strong (1919) stated that syphilis was very rare indeed in the native population of Papua. Ford (1939) made a survey of venereal disease in the Trobriand Islands in 1939, and did not find any cases of syphilis in an examination of 5400 persons. He found cases of yaws which resembled syphilis, but was able to obtain a history of the earlier stages of yaws in these patients. It is interesting to note that Ford found no case of ulcerating granuloma of the pudenda during this visit to the Trobriands.

From the evidence available it is not possible to make a definite statement on the accuracy of Bellamy's clinical diagnosis of syphilis. If syphilis was present in the Trobriand Islands, it appears to have died out; but there is the earlier evidence of the somewhat rare occurrence of a condition which, according to expert medical opinion of the time, was syphilis. It would be reasonable to suppose that some, at least, of Bellamy's cases were indeed syphilis. It is doubtful whether even modern evidence would allow a categorical statement to be made on the presence or absence of syphilis in Papua-New Guinea.

Ford's survey in 1939 revealed 16 cases confirmed as gonorrhoea by microscopic examination. In addition, there were 214 males whose urine in the two-glass test was found to contain threads but in whom no gonococci could be demonstrated. Ford remarked that Bellamy had probably diagnosed such cases as gonorrhoea.

There is little indication of the methods employed by Bellamy in the treatment of the various venereal diseases. Strong stated that in 1918 the usual treatment of venereal ulcerations in Papua consisted either in the administration of large doses of potassium iodide (100 to 200 grains per day) and the local application of calomel, or early operation, repeated if necessary. He reported that operation was especially preferred at Samarai, so that it can be inferred that Bellamy mainly used medical treatment for this condition. In one annual report Bellamy recorded ten cases of venereal disease in which surgical treatment was required. Bellamy noted that chancreoid ulceration was frequently very difficult to treat, especially in some cases in which there was a suspicion of syphilis. When there was a possibility of syphilis he used antisyphilitic treatment in addition to the local chancreoid remedies, generally with great improvement and speedy healing of the ulcers. "Salvarsan" was put on the market in December, 1910, but the arsenicals were not in general use in Papua—for the treatment of yaws—until much later; thus, in the earlier

days of the special hospital at Losuia, Bellamy probably treated the conditions diagnosed as syphilis with mercury and potassium iodide.

There is also little to indicate Bellamy's method of treatment for gonorrhoea. Barton, in his letter offering the position at the Trobriands to Bellamy, stated that the necessary drugs and instruments had been ordered for the hospital. These instruments may have included urethral syringes and sounds. Ford (1955), when camped at one of the Trobriand villages in 1939, "heard a bright and tuneful song on the vicissitudes of an unfortunate lover who had received Bellamy's professional attention. It



FIGURE VIII.

Coral jetty at Losuia, Trobriand Islands, commenced by Moreton in 1905 and extended by Bellamy.

was accompanied by actions which made the theme quite clear, and caused great amusement. The final line in each verse indicated syringing of the tender part, accompanied by loud repetition of 'pump-pump-pump' (apparently a Trobriand word!). Recent inquiry indicates that this song and dance may have referred to treatment given by Mr. Whitehouse, who was placed in charge of the special hospital at Losuia after Bellamy returned from the war. The dance was called *sina bara*.



FIGURE IX.

The town of Port Moresby after the first World War; the European hospital is at the top left.

The results of Bellamy's efforts to combat venereal disease are apparent from Table I, which shows the incidence of these diseases over the period from 1908 to 1915. During this time the percentage of the population affected fell from 5.2 to 1.4. Bellamy himself (1908) said that he did not believe venereal diseases would ever be eradicated, but he was certain that they could be and were being effectively controlled. There are no figures on the incidence of the disease in the area before the hospital commenced to function in 1905; but Bellamy stated in 1906 that 10% of the total population were affected by venereal disease, and as many as 20% in some of the villages (Bellamy, 1925).

After his return from the war and after his graduation in medicine, Bellamy visited the Trobriand Islands in February, 1919, and examined over 1000 of the native inhabitants. He

found that between 4% and 5% were suffering from genital sores of one kind or another, compared with a figure of just over 1% for this group prior to his going on active service leave. Mr. E. Whitehouse was put in charge of the special hospital at Losuia, and by following Bellamy's method of regular inspection of all the native people and treatment of the affected persons found, soon reduced the incidence of venereal infections to 1%.

Strong (1916), commenting on the reduction in the amount of venereal disease in the Trobriand Islands, regretted that the resources of Papua did not permit of a medical officer's being stationed with each group of 10,000 natives. Later (Strong, 1921) he stated that it was fair to assume that the result of the Government's having stationed an officer on the Trobriands was that there was not more than one-quarter of the venereal disease which there would have been in his absence. Indeed, the reduction in the amount of venereal disease was probably much greater.

The Lieutenant-Governor of Papua, J. H. P. Murray, paid tribute to the results of Bellamy's work in the control of venereal disease in an island group where, as Bellamy himself was well aware, sexual intercourse was very free between the unmarried and began at a very early age. Murray (1913) stated that "by degrees the tact and perseverance of . . . Bellamy, added to his successful treatment of the cases which he undertook, triumphed over the fears of the sufferers".

Ford (1939) found no difficulty at all in gathering the people for examination, "apparently the result of years of medical work on the island by Dr. R. L. Bellamy, ARM . . . the occasion was accepted as a gala event". Ford went on to say that the education of the Trobriand Islanders in regard to venereal infection was well founded; men and women presented themselves willingly for examination without suspicion or distrust. This desirable condition was attributed by Ford to the early labours of Bellamy and was a "monument to his understanding and assiduous work".

It is fitting to close this chapter on venereal disease with a statement by Bellamy (1920) on the method of control of venereal disease in Papua, made when he was acting as Chief Medical Officer, 1919-1920:

The longer my experience of venereal in Papua, the more convinced I become that the only method which will reduce this menace to reasonable proportions is for a census to be taken house by house, village by village, of every man, woman and child, and for every man, woman and child to be inspected by a qualified medical man at least once in every twelve months. I strongly suspect that, in many cases, we only get into touch with the very bad cases, those who are obviously so sick that everybody in the village knows about them, but we fail to get those earlier cases who walk about like fit men and women and yet who are suffering from venereal, and are the cause of that insidious spread of the disease which it is our duty to prevent. So long as such foci for the dissemination of the disease are able to escape detection, so long will our efforts fall short of the success we desire to attain.

Epidemiology

Bellamy was never content with only the diagnosis and treatment of the diseases he encountered. His inquiring mind always sought information on the incidence and method of spread of disease, and the circumstances which influenced the natural history of disease. His interest in the behaviour of native peoples and his ability to speak with them helped him considerably in his search for epidemiological detail with which his note-books must have been packed full. Some of his observations on various diseases are presented here, and it should be remembered that they were mostly made in the earlier years of this century.

It has been mentioned, in dealing with Bellamy's work on the control of venereal disease, that he recorded the sexual and marriage habits of the Trobriand people and their marriage pattern according to clans, and his method of attack on the problem of control automatically included observations of the incidence of the disease.

Bellamy appears to have been the first, since the early observations of MacGregor, to diagnose leprosy in the Territory. Beaumont, the Chief Medical Officer (1908), apparently was unaware of MacGregor's reporting of this disease and said, with reference to Bellamy's diagnosis of leprosy, that he did not know of any cases in the rest of the Territory, although a suspected case had been seen some years before in Port Moresby, but it was concluded not to be leprosy. However, Bellamy found that the Trobriand people had a word for the disease—*Kaikwaigwa*—and that it must therefore have been present for some time. He examined patients with leprosy with Breinl during the latter's visit to the Trobriands, and was able

to provide the information that the cases were apparently confined to one village in the group.

After some years' residence in the Trobriand Islands, Bellamy noted that a very wet and prolonged north-west season resulted in an increased amount of malaria. He said that these conditions were favourable for an increase in the number of anopheline mosquitoes. He described some of the clinical features of malignant malaria, and this description is one of the very few which have been written on the clinical features of malaria in the New Guinea native. He found that in a bad season as many as 50% of the inhabitants of a village might suffer from a malarial attack. The native people soon came to recognize the value of quinine.

Bellamy had some comment to make on the accident patients whom he examined in the Trobriand Islands and admitted to the special hospital for treatment. He found it remarkable that so few accidents occurred in a population of approximately 10,000 people. The types of injuries he saw were simple fractures, wounds from tomahawk and knife—sometimes accidental, sometimes resulting from a quarrel usually over a garden or a woman—and occasionally a spear wound. He also treated some patients with animal or vegetable poisoning. In a letter written in April, 1915, Bellamy described a shark attack upon one of the Trobriand natives, who had a leg badly mauled. The patient "had dived down into about 2½ fathoms and was coming up to the surface when the shark, seeing the white of his underfeet, seizing him . . . it was certainly the flash of the soles of his feet which attracted the shark". He thought that the victim of this attack would survive.

Bellamy described a dysentery epidemic which occurred in April, 1911, at the change from the north-west to the south-east season. He first pointed out that at this particular time of the year the new season's yams were always taken up from the gardens and there was always a good deal of "catarrhal inflammation of the bowels" about. The natives explained that this was due to the change to the new food. Bellamy thought that they were probably right; but, at any rate, it was an annual occurrence. This epidemic of dysentery commenced in a coastal village. He found that only one-half of the village was affected and the patients drew their water from a stream at one end of the village. Those people from the unaffected part of the village drew their water from a waterhole inland, which was quite separate from the stream. Bellamy tabooed the stream, isolated the patients on a beach site away from the village, and isolated the village. However, the disease had been spread by people from another village who had attended a funeral in the stricken village. With the constant trading which went on it was impossible to isolate the affected area, and the disease spread, resulting in the death of 140 native people, of whom 118 were infants and children. The disease mainly attacked the very old and the very young and was fatal for infants. Bellamy had difficulty in treating the patients, as the parents persisted in giving solid food to the sick children. Bellamy attributed the commencement of the outbreak to the eating of a sick pig which had a discharge from the bowels. If his deduction was correct, a salmonella infection may perhaps have been expected rather than bacillary dysentery.

With regard to respiratory diseases, Bellamy considered that all the native people throughout Papua had a predisposition to diseases of the chest. He stated that tuberculosis was especially found amongst those who lived near centres of European population. Bellamy thought that there appeared to be some evidence that the more nearly the native tried to imitate the white man in his clothing and food, the more liable he was to tuberculous infection.

He agreed with Strong that the cause of New Guinea mouth, an ulcerating condition of the buccal mucous membrane, was a form of scurvy and attributable to a deficiency or complete absence of a "vitamine" from the diet; thus it was comparable with beriberi and rickets, and a preventable disease. Recent medical opinion admits a nutritional basis in the aetiology of New Guinea mouth, but adds a bacterial factor as well.

Some comments on the European in the tropics were made by Bellamy after he had lived in Papua for over fifteen years:

I have seen the proverbial heavy drinker and the pronounced teetotaler fall equal victims to the climate, so that I cannot accept the theory that total abstinence from the various forms of alcoholic beverage is all that is necessary to insure freedom from certain pathological effects which result from prolonged residence in a malarial country.

I have seen fair complexioned men and women weather successfully repeated attacks of malaria, while Europeans of darker-skinned type have gone under. There have been, of course, exceptions here and there

but allowing for this, there is something more than accident or coincidence in the observed results of type susceptibility to the malarial and other conditions of a tropical country.

And while there is much that must be accepted uncomplainingly as unavoidable in the nature of things by those whose business it is to carry on in Papua, there is also much which the people can do themselves to deserve, if not to insure, that degree of relative freedom from the ills which dog tropical footsteps. In nothing is this more evident than in the matter of diet.

Vital Statistics.

In so far as vital statistics is a tool of the epidemiologist, some of Bellamy's work, which could be classed under the present heading, has already been described. There remains for discussion the important subject of population statistics, in which he was keenly interested.

In the first few years of the century Moreton, the Resident Magistrate who visited the Trobriand Islands, and the Reverend M. K. Gilmour attempted a count of the number of people who lived in these islands. Their count in 1904-1905 was 10,408 (Trobriands and Lausancay) and 798 in Kitava. Moreton (1905) expressed the opinion that the population was undoubtedly decreasing, owing to famine and disease.

When Bellamy found that the cases of venereal disease were becoming fewer, he suspected that he was not seeing all the cases which occurred. He set about his systematic examination of all the people on the Trobriand Islands in 1908. All names were entered in a book, and his nominal roll was comparable to a census of the Trobriand people. After a few years he had recorded the names of over 7000 people, and in 1913 he commenced, with the aid of the village constables, the recording of all births and deaths by months. Bellamy stated that the reason for this registration of births and deaths was that the question of the dying out or otherwise of the Papuan had an important bearing on the development of the Territory.

In his first period of observation, 1913-1914, there were 325 births and 266 deaths, with corresponding annual rates of 38.2 and 31.2 per thousand. Bellamy compared these figures with some from other countries as quoted in a recent "Whitaker's Almanack". He stated that the birth register was of too recent origin to allow any definite conclusions to be drawn. Bellamy noted that the Trobriand Islander did not usually care for large families and a twin birth annoyed him: "People will think my wife is a pig, and is bringing forth a litter." This is an attitude which is, of course, not confined to the Trobriands. The ratio of male to female births was 167 to 158 (106 to 100), and Bellamy commented that female infanticide had occurred in the past and he was not certain that it had disappeared. He hoped that, if it was still practised, the birth register would check infanticides, as there were 1412 unmarried males and only 856 unmarried females. Of the deaths recorded in that year, 37.9% were those of infants and young children.

It was not until two years later that a similar record was made elsewhere in Papua, and this was by Strong (1919) in the villages at Port Moresby with a population of 1670 (Bellamy, 1920).

In the year 1914 Bellamy compared the birth and death rates in the Trobriands with the figures for Sydney, and noted that the death rate was comparatively high and that the high birth rate was abundantly needed. He commented with interest upon the large number of births in the month of May in both 1913 and 1914. Counting back to the date of conception he arrived at September, which was the height of the Trobriand village dancing. The native food crops were by then safely garnered, food was abundant, pigs were killed and there was general rejoicing. Bellamy asked: "Is September or its neighbourhood the breeding season for the Trobriand Islanders?"

The register of births and deaths was continued when Bellamy left the Trobriands Islands to serve in the first World War, but possibly not so efficiently until Whitehouse arrived in 1920. When he was Acting Chief Medical Officer in 1919, Bellamy (1920) stated that the only two areas where birth and death rates could be given for Papua were in the Trobriand Islands and the small native community in the neighbourhood of Port Moresby.

This interest in population remained with Bellamy and apparently received some stimulus during the course in tropical medicine in Townsville, for, immediately after his return to Papua, he proceeded to the Trobriand Islands, and a few months later wrote his "Enquiry into Vital Statistics of the Trobriand Group". A copy of this report has recently been located in

the old files at Losuia by the Assistant District Officer, Mr. L. Doolan. The report dealt with the period from July 1, 1919, to June 30, 1926. Bellamy noted that the Birth and Death Register had not been kept in the period 1915-1919, and that there was also a gap in 1924 when Whitehouse had been on leave. He found that the population had begun slowly to decrease over that period, in contrast with the slow increase he had observed during the time when he was Resident Magistrate.

The report contains a careful analysis of the factors concerned in this change, and concludes with a comment upon the striking differences in conditions in the Trobriands in 1926 compared with those existing when Bellamy left to go to the war. Notable amongst these changes was a considerable loss in prestige of the chiefs, which had an effect upon the whole village life and especially upon gardening and, therefore, food supplies. Bellamy also considered that Kiriwina was "suffering from too much missionary". He considered that the position in the Trobriands could be summed up in the statement of one Trobriander: "Taubada, if I don't pay my tax I go to gaol, if I don't give the missionary money I go to the 'big fire'."



FIGURE X.

Mountain torrent (Kumusi river) in the Northern Division of Papua, where Bellamy spent much of his time as Travelling Medical Officer.

This report is a valuable commentary upon change in a Papuan community as well as a technical discussion of the factors affecting its population numbers.

Food Supplies.

Early in the century a move was being made throughout British New Guinea to persuade the native people to plant coconuts (for example, Campbell, 1905). In 1910 Bellamy commenced planting in the Trobriands. His methods of securing cooperation will be described elsewhere; but the results were that four years later 120,694 nuts had been planted. This move ensured that coconuts would be available for everyone—chief and commoner alike. In addition to being a supplementary and reserve food, the coconut provided copra, which could be sold or traded by the people.

It was Bellamy's policy to use locally grown yams, taitu, taro, etc., for feeding the patients in the special hospital, so he had every reason to be interested in the condition of the local gardens. He did not use rice, which was not grown on the Trobriands. In addition, he offered advice when crops failed, suggesting what could be grown quickly to avert starvation—for

example, potatoes and maize—and obtained the seed from Port Moresby.

As the amount of food available fluctuated considerably, Bellamy supervised a station garden worked by convalescents, and although the land was near the coast and not very well suited for gardening, he was able in one year to produce—at the height of a general food shortage—sufficient to feed for six weeks 80 to 85 people in the hospital and on the station. He experimented with the growing of English vegetables, and reported that, under favourable rain conditions, cucumbers, tomatoes, radishes, turnips, French beans, lettuce, parsley, watermelon, mint and pumpkins did well on the Trobriand Islands. He had, in addition to these vegetables, 150 mandarin and orange trees. To accommodate reserves of food he had eight large food houses with a capacity of 70 tons. He kept ducks for his own table, and sent tomatoes to the mission staff from his own garden.

Mrs. Bellamy stated that her husband used to plant citrus and other useful trees wherever he went in Papua. She maintained that the fruit enjoyed by the troops at Kokoda during the War of 1939 to 1945 came from trees which had been planted by Bellamy in the early years of the century. On the Trobriand Islands she relates that Bellamy imported pigs—possibly the Berkshire referred to by Rentoul (1932)—to improve the breeding of the village pigs. From the new litters he claimed a certain number, which were then sent to other villages.

As part of his magisterial duties Bellamy supervised the pearl fishing in the Trobriand lagoon. This trade was governed by regulations designed to prevent the fishing out of the *lapi*—the edible mollusc *Margaritifera vulgaris*—which formed part of the Trobriand Islanders' food supply.

Bellamy commented on the probability of a change to European diet rendering the Papuan susceptible to tuberculosis, and this observation, together with his feeding of patients and station personnel on locally grown foodstuffs, leads to the conclusion that he considered that the foods the native people had always eaten were the most suitable for them. In this regard it is interesting to note that the nutrition survey made in 1947 (Hipsley and Clements, undated) found the diet of the Kavataria people (Trobriand Islands) adequate, except perhaps for some deficiency in calcium. In other areas, of course, the results were different.

Bellamy had decided ideas about the diet of the European in the tropics. He wrote:

One continually finds people adopting the same routine diet which pleased and, no doubt, suited them in a colder climate—bacon persistently for breakfast, blood-heating cereals, meat three times a day, mostly tinned, but still meat; port popular, because it is easily procurable, as it is grown locally; and half a dozen other unsuitable things allowable elsewhere, but not here.

It is any wonder, therefore, that there comes a breakdown with gastric and hepatic symptoms, complicated with the inevitable attack of malaria?

Public Health Administration.

Bellamy's introduction to medical administration came with his first appointment to the special hospital at Losuia in 1905. Here he had to build up the organization of this hospital from the beginning, train the staff and supervise food and medical supplies, as well as carrying out his professional work. His reports indicate that this was done well and effectively.

On his return from the War of 1914 to 1918 with his medical diploma, he was very soon called upon to act as Chief Medical Officer while Strong was absent on leave for one year. This period lasted from April, 1919, to May, 1920. The position also included the duties of Chief Health Officer and Chief Quarantine Officer (Strong, 1921). Bellamy's report for the Medical Department for the year ending June 30, 1919, indicates that the department ran smoothly under his care. All the work reported is carefully identified with the officer who was responsible. That he was capable of making effective changes is shown by the posting of Mr. Whitehouse to the Trobriands where, apparently, Bellamy was not satisfied with the way in which medical affairs were being managed.

During this period the quarantine station on an island near Port Moresby—Dauko—was converted from a collection of primitive buildings to a well-ordered station. Haste was necessary in its preparation, as news had been received of the outbreak of pneumonic influenza in Australia and Samoa. Bellamy stated that the Port Quarantine Officer, Dr. B. C. N. O'Reilly, supervised the building arrangements.

Bellamy's comments upon the reports of the officers of the department show that he was well aware of the problems of public health throughout Papua and was familiar with their historical background. He realized that wholesale death would occur amongst the native population if influenza entered the country, and therefore closed Daru, in the west, as a port of entry to the Territory; ships from Thursday Island had first to call at Port Moresby. No ship was allowed to enter any Papuan port until ten days after her last contact with an Australian port. Bellamy noted that Papua was one of the few countries in the world which had, so far, escaped this scourge.

He gave an historical outline of the work on hookworm infestation in Papua, and described the work of Dr. Thomson during the period under review. Bellamy's interest in the epidemiology of this disease led him to have all patients admitted to the Port Moresby native hospital examined by Mr. A. S. Graham for hookworm ova.

He commented on two deaths from blackwater fever which occurred in the Port Moresby native hospital, and noted that the victims were Polynesians, not Papuans. Bellamy was apparently aware that this disease is very rare in the native people of Papua.

In 1924 also, Bellamy acted as Chief Medical Officer. His annual report on the Medical and Health Department showed the same features as the first, with some emphasis on the costs of maintaining the hospitals.

However proficient he was in carrying out the duties of Chief Medical Officer when called upon to do so, Bellamy preferred the life of the Travelling Medical Officer, which took him amongst the village peoples of Papua and away from Port Moresby. Among the native people he was content to do what he could in improving their health, observing their behaviour and adding to his knowledge of the Papuan people by first-hand contact with them. For this reason, and also because Strong had the senior qualifications, there was no question of Bellamy's being appointed Chief Medical Officer of the Territory, which appointment was given to Strong in the year 1921–1922. It is interesting to recall that Strong was first appointed in Papua two months earlier than Bellamy in 1904, but remained for many years in the magisterial division before transferring to the Medical Department.

Other Public Health Activities.

The station and hospital at Losuia were judged to be very comfortable and creditable by Symons (1912), and ornamental shrubs, such as crotons and hibiscus, were planted around. Bellamy, with his well-ordered station, was in a position to insist on the villages being kept clean and tidy. He considered that this would lead to the better general health of the inhabitants. In one case he moved the coastal village of Teiava from an exceptionally unhealthy site to one which was more elevated. This must have been an unprecedented achievement, and it certainly was a revolutionary approach to village sanitation in Papua in 1910.

The year 1913 was one of those in which the north-west wet season was prolonged, with the inevitable and considerable increase in the number of patients with malaria seeking treatment at the special hospital. Prisoners and patients at the station were also sufferers. Bellamy reported that he had carried out an energetic anti-mosquito campaign, destroying every possible nidus, and stringent measures were taken in the villages. This, too, was a most unusual approach to malaria control in a territory where nothing had previously been done in the control of rural malaria. Strong, commenting upon the report of Bellamy's mosquito control work, suggested that complete extermination of mosquitoes was an impracticable ideal away from the main centres of European population.

There is little record of the work done by Bellamy in his later years when he was a Travelling Government Medical Officer—records at Port Moresby were lost during the second World War. In one year he gave 4000 treatments for hookworm infestation in the Eastern Division as part of the hookworm campaign initiated by Lambert, whom Bellamy had met in the Trobriand Islands. Mrs. Bellamy spoke of the lining up of Trobriand villagers for injections, which were probably for the treatment of yaws.

It can easily be supposed that he applied the same diligence and thoroughness as characterized his work in the Trobriand Islands, that always he was interested in the reason behind the observed detail, and that every observation was carefully written down in his note-book, probably with some whimsical comment.

BREAST CHANGES IN THE MALE AND FEMALE WITH CHLORPROMAZINE OR RESERPINE THERAPY.

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WITH any new drug, frequent obvious or dangerous complications receive early attention; other side reactions are described later. Chlorpromazine and reserpine have proved no exception, and recently breast changes have been described after the use of each.

Chlorpromazine.

Enlargement of one or both breasts with the secretion of a milk-like fluid during chlorpromazine therapy has been reported. The swelling is diffuse and is not associated with cyst formation or tenderness; the areolae may become widened. The secretion is variously described as milk or colostrum or as resembling "witch's milk". The amount may be copious, but is usually minimal, and then perceptible only on expression. Lactation is most common in younger patients when high dosages are being used; the duration of treatment seems of less importance, and with continuance the secretion may cease while the breasts remain enlarged. The urine when tested does not contain lactose. It is stated that the menses are sometimes disturbed; but such irregularities are common in patients with nervous disorders (Kinross-Wright, 1955; Moyer *et al*, 1955; Cohen, 1955, 1956; Morgan, 1956; Tenenblatt and Spango, 1956).

The incidence of the complication in the different series has been from 1% up to 80%. Ayd (1955) makes the following statement:

I have found approximately 80 per cent. of women receiving this drug lactate. In a few cases the lactate oozes from the breast, but in the majority of cases the lactate must be expressed. The volume varies from a few drops to as much as 20 or 30 c.cm.

A possible explanation for these differences is that in some series only the obvious cases, in which the secretion was copious, had been observed and recorded, whilst in others expression had been attempted. It must also be borne in mind that while idiopathic galactorrhoea is very rare, a watery, whitish fluid occasionally can be expressed from the breasts of parous women. As no reference could be found to the frequency of this occurrence, the breasts of 60 parous women were examined; 41 of these were premenopausal and 19 were post-menopausal. None was receiving chlorpromazine, reserpine or allied drug, and none had recently been pregnant. In none could fluid be expressed (Table I).

Interest in this complication of chlorpromazine therapy was aroused when a young nulliparous woman at the Royal Park Receiving House was found to be lactating. Her history is as follows:

CASE I.—Miss A. is an intelligent, but immature and dependent, nulliparous woman, aged twenty-two years. She has been an in-patient in mental hospitals on various occasions since 1954. She was a borderline psychotic of a mixed hysteroid and schizophrenic type, but after a leucotomy in 1955 her thinking became more obviously schizophrenic. In 1956, while taking 300 milligrammes of chlorpromazine a day, she began to secrete a milk-like fluid very freely from her breasts. Her menstrual periods were regular, and there was no clinical evidence of other endocrine disorder. At first the lactation was thought to be possibly a psychosomatic disorder, as one manifestation of her immaturity was her preoccupation with a favourite teddy bear, which she would fondle by day and cuddle by night with maternal solicitude. However, at this time a report of lactation with chlorpromazine was seen, and its administration was stopped. When she was examined a month later lactation had ceased. Later it became necessary to resume the treatment, and within three weeks the fluid was again present.

This was the first such case observed in either the Royal Park Receiving House or the Kew Mental Hospital, in both of which many women had received large doses of the drug for relatively long periods. In neither hospital had routine attempts at expression been made. With this in view, 69 other women who had been receiving chlorpromazine in doses varying from 75 to 600 milligrammes a

TABLE I.

Control Series. No Patient was Taking Chlorpromazine, Reserpine or Allied Drug and None had Recently been Pregnant. In None was Milk Present.

Age (Years).	Nulliparous.	Parous.	Total.
15 to 50	57	41	98
Over 50	15	19	34
Total	72	60	132

day for periods of over one month were examined. None with a post-partum psychosis was included. As adequate menstrual histories could not always be obtained, the patients were grouped according to age. Including the first patient, there were 41 aged less than fifty years and 29 older ones. Milk was present in seven patients, all of whom were from the younger age group, and all of whom were receiving large doses (Table II).

TABLE II.

Incidence of Lactation in Female Patients Taking Chlorpromazine, Reserpine or "Pacatal".

Drug.	Age (Years).	Number Examined.	Number Lactating.
Chlorpromazine.	15 to 50	41	7
	Over 50	29	—
	Total	70	7
Reserpine.	15 to 50	22	2
	Over 50	4	—
	Total	26	2
Chlorpromazine with reserpine.	15 to 50	1	1
	Over 50	1	—
	Total	2	1
"Pacatal."	15 to 50	9	—
	Over 50	2	—
	Total	11	—

CASE II.—Miss B., a nulliparous girl, aged fifteen years, had been suffering from schizophrenia with delusions and hallucinations for six months. She was admitted to a mental hospital after an attempted suicide. She was given chlorpromazine, 300 milligrammes daily, for one month, when this was combined with full-coma insulin therapy. When she was examined two weeks later, a small amount of fluid could be expressed from each breast. On biochemical examination of the fluid, the protein appeared in type and content—3.5%—to be typical of that in colostrum. However, the fluid did not coagulate on being boiled, and the microscopic appearance was more similar to that of breast milk. The quantity obtained was not sufficient for further tests. The Friedman Lapham test produced a negative result. Her urine contained a reducing substance that was shown to be glucose by the osazone test; this is not unusual in patients receiving full-coma insulin therapy. No lactose was detected.

CASE III.—Miss C. is a nulliparous girl, aged nineteen years, with florid paranoid schizophrenia. She was given chlorpromazine, 300 milligrammes daily. When she was examined six weeks later, a small amount of white fluid could be expressed from each breast.

CASE IV.—Miss D., a nulliparous girl, aged twenty years, has schizophrenia with obsessive features. After she had received chlorpromazine, 450 milligrammes a day, for one month, a small amount of white fluid could be expressed from each breast.

CASE V.—Mrs. E. is aged thirty-six years. Her youngest child is aged five years. She received 300 milligrammes of chlorpromazine daily for a reactive depressive state. A few drops of milk could be expressed from the right breast.

CASE VI.—Mrs. F., aged thirty-six years, is a Hungarian who has been in Australia for six years. Her youngest child is aged four and a half years. She has had several episodes of paranoid schizophrenia. While taking 150 milligrammes of chlorpromazine a day, she was admitted to a mental hospital, threatening suicide, as she believed she was pregnant. She had discovered some milk in her breasts. This was evident only on expression. The uterus was small, and the result of the Aschheim-Zondek test was negative. There was no lactose in her urine.

CASE VII.—Miss G., a nulliparous woman, aged thirty-six years, is an in-patient in a mental hospital. She is a chronic schizophrenic with hysterical and hypochondriac symptoms. Since June, 1956, she has received chlorpromazine, 225 milligrammes a day. In November, a symptomless lump was found in the right breast. A few drops of milk could be expressed. The left breast appeared normal. At operation the mass shelled out easily. This was irregularly ovoid in shape, firm in texture and encapsulated, and measured 2.0 by 1.5 centimetres. On microscopic examination, the nodule was an intracanalicular fibroadenoma, in which the duct epithelium showed several layers of regular cells and the stroma was abundant and mucoid in type. A small amount of surrounding breast tissue contained numerous ducts arranged in lobular form. The epithelium was regular, and there was no evidence, either here or in the nodule, of secreting epithelium. The condition was a lobular hyperplasia ("chronic mastitis"), the result of some chemical (presumably hormonal) stimulation. The presence of the fibroadenoma was presumably fortuitous and not related to the therapy; but there was in the tissue clear evidence also of stimulation.

These findings help to explain the difference in the incidence of this complication in the published reports. In the present series, had expression not been attempted, the incidence would have been 1.4% instead of 10%. However, many of the patients examined were in the older age groups in which the reaction does not occur, and some of the younger ones were receiving relatively small doses. There were 20 patients aged between fifteen and thirty-six years who were receiving more than 200 milligrammes of chlorpromazine a day. Six of the cases of lactation were from this group, so that here the incidence was 30%. Furthermore, the majority of these patients were examined only once, and it is thought that had this examination been repeated the incidence would have been still higher. Thus it is likely that these changes will be frequent only in severely disturbed young psychotic women in mental hospitals, and that they will not be seen in psychoneurotic patients in general practice who receive relatively small doses.

Reserpine.

Similar breast changes to those observed in patients receiving chlorpromazine have been described during reserpine therapy. Ayd (1956) observed painful swelling of the breasts, and Platt and Sears (1956) found three cases of lactation amongst 54 hypertensive patients; the three lactating patients were aged twenty-nine, thirty-eight and forty-nine years respectively. The changes were not related to parity. In one case the fluid was examined biochemically and microscopically and was regarded as being normal human milk, although the protein content was higher than average. The secretion may cease even if the treatment is continued.

With these statements in mind, the breasts of 26 women who had been receiving reserpine in doses of from 0.75 to 5.0 milligrammes daily for periods of over one month were examined. Twenty-two of these were aged under fifty years, while four were older. Milk was present in small amounts in two cases (Table II). Both patients are nulliparous. They are congenital mental defectives who have been in-patients in mental hospitals for many years. Their respective ages are twenty-six and thirty-nine years. Both had been receiving three milligrammes of reserpine daily.

Two other patients were examined who were taking both chlorpromazine and reserpine. In one, aged fifty-eight years, no milk was found. In the other, aged forty-two years, a small amount could be expressed from one

breast. She is a nulliparous, deteriorated epileptic with schizophrenia, who had been receiving 300 milligrammes of chlorpromazine daily for two weeks and three milligrammes of reserpine daily for two months.

Gynecomastia with Reserpine.

No record of gynecomastia occurring with either chlorpromazine or reserpine was found. The next case is therefore of interest.

CASE VIII.—Mr. Z. is a business executive, aged fifty-six years. During October, 1955, he became emotionally disturbed and was unable to work. Some of his symptoms were those of cerebral arteriosclerosis; but others were functional and related to past and present emotional problems. He received psychotherapy, and in addition was given reserpine, 0.5 milligramme a day. In April, 1956, this was reduced to 0.25 milligramme a day. In July, after taking reserpine for nine months, he noticed that his breasts were enlarging and that the nipples were tender. He was examined two months later, and in each breast there was a plaque of firm, glandular tissue, about 8.0 centimetres in diameter and 1.5 centimetres in thickness. No fluid could be expressed. He took no more reserpine. A month later the breasts were smaller and much softer, and any enlargement appeared to be due to fat alone. By November the swelling was hardly perceptible, although the nipples were still slightly tender. No special investigations were made, but no other cause of gynecomastia was found on clinical examination. He was not taking any oestrogen.

This patient was not treated by the Mental Hygiene Department; but no case of gynecomastia was observed in the Royal Park Receiving House or the Kew Mental Hospital among patients receiving either chlorpromazine or reserpine.

Hypertension.

Many hypertensive females take small amounts of reserpine for long periods. It is unlikely that breast changes will be frequent in these, as most are in older age groups. Here, too, the doses are small. In the present series, most patients examined were taking amounts far in excess of these, so it was not possible to conclude that here lactation is a function of dosage rather than of duration of treatment, as was done with chlorpromazine. It is to be noted, however, that the patient with gynecomastia was aged fifty-six years, and that his dosage, while small, was continued for a long period.

Parity.

As lactation with chlorpromazine or reserpine is not related to parity, 72 nulliparous women, who were not receiving these or related drugs, were examined. In none could fluid be expressed (Table I).

"Pacatal."

Eleven women receiving "Pacatal" in doses of 50 to 150 milligrammes a day were examined. All were aged over thirty-three years. In none was milk present (Table II).

Discussion.

Enlargement of the breasts can be caused by oestrogens, and these, which are secreted in both males and females, are destroyed in the liver. If there is liver disease this may not occur, and gynecomastia thus can result. Chlorpromazine can cause liver damage; but the patient described as having gynecomastia was receiving reserpine, which is not regarded as being a hepatotoxic substance. None of the patients described was jaundiced.

While oestrogens cause proliferation of the ducts and enlargement of the breasts, physiological lactation is the result of the secretion of prolactin, and possibly of other hormones, by the adenohypophysis. Furthermore, prolactin initiates and maintains the secretory activity of the corpus luteum, and while progesterone alone produces no changes in the immature breast, conspicuous acinar development will occur if it is combined with oestrogen.

The mode of action of either chlorpromazine or reserpine is not clearly known; but certain effects of both are presumed to be mediated through the hypothalamus. Thus

both indirectly could affect the adenohypophysis, which then could initiate the breast changes. There also remains the possibility that chlorpromazine and reserpine can themselves act as if they were hormones and directly affect the breast.

Some believe that schizophrenia can best be regarded as being primarily a metabolic disease. In this survey, most of the patients who lactated with chlorpromazine treatment were schizophrenic women, while those whose breast changes occurred with reserpine were not.

Certain effects of reserpine may be due to the release of serotonin in some undefined part of the brain (Shore *et al.*, 1955); chlorpromazine does not act thus. Serotonin is an oxidation product of tryptamine, and it is believed that one of its functions is concerned with nerve metabolism. It is antagonized by the hallucinogen *d*-lysergic acid diethylamide (LSD). LSD, serotonin and reserpine have structural similarities. The mental changes caused by hallucinogens such as LSD may be "the result of a serotonin-deficiency which they induce in the brain. If this be true, then the naturally occurring mental disorders—for example, schizophrenia—which are mimicked by these drugs, may be pictured as being the result of a cerebral serotonin-deficiency arising from metabolic failure rather than from drug action" (Woolley and Shaw, 1954a, 1954b).

As neither the mode of action of these drugs nor the aetiology of schizophrenia is known, any discussion must be conjectural; but it is thought that these isolated findings are suggestive and may prove to be significant clues to a future understanding of some disordered mental processes.

Summary.

Enlargement of the breasts in both sexes and secretion of a milk-like fluid in females during chlorpromazine or reserpine therapy are described.

Sixty-six women receiving chlorpromazine were examined. A milk-like fluid could be expressed from the breasts of seven.

Twenty-six women receiving reserpine were examined. A milk-like fluid was present in the breasts of two.

A case of gynecomastia occurring with reserpine therapy is described.

Eleven women receiving "Pacatal" were examined. None was lactating.

All patients who lactated were below the age of forty years, and all were receiving large doses of either chlorpromazine or reserpine; the complication is therefore most likely to be seen in severely disturbed young psychotic women. The changes are not related to parity. The secretion when examined had features of both milk and colostrum, and the urine when tested did not contain lactose.

The manner in which these changes may be brought about is discussed.

Acknowledgement.

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PSYCHIATRIC CONSEQUENCES OF RAUWOLFIA THERAPY.

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OVER the past five years the medical profession has been assailed with a spate of new therapeutic drugs. It is now becoming evident that many of these are to be used prudently and their side effects to be cautiously heeded.

The serious psychiatric consequences of rauwolfia therapy as used in the management of hypertension and other ailments should be brought to notice.

During the past eighteen months Dr. S. J. Minogue and I have seen 32 patients who have developed a peculiar insanity whilst having their blood pressure treated with rauwolfia extracts. The drug manufacturers admit there is some 5% risk.

This is a serious problem. A normal person, without a previous history of neurosis or insanity in his or her lifetime, is launched on a course of "blue pills" for his "pressure" and winds up with a psychosis.

The position is even more serious than this, for once the psychiatric complications are manifest, it is not just a case of stopping the tablets. The patient is left with a wretched mental state that may take a full three to six months to clear up.

Three of the most determined cases of suicide that I have recently encountered (both wrists and the throat deeply cut) have been in patients taking rauwolfia preparations.

A storekeeper and his wife from a country town were examined; he had a reserpine "depressed" state. When his condition was explained to him, he and his wife suddenly recalled that two people in the district had suicided by drowning in the local river, and they had been taking the tablets. The conversation was steered into other channels.

What, then, are the incipient or early phases of this rauwolfia reaction? The history is a typical one. A patient is discovered to have moderately severe hypertension. He is told this, even to the exact figures of his blood pressure, and he is started on a course of rauwolfia—for example, "Serpasil", 0.25 milligramme three times a day. Within some weeks his blood pressure has fallen; the patient knows the exact amount of the fall—the emphasis of both physician and patient has settled on a reading on a sphygmomanometer. There is a feeling of satisfaction in both parties.

So the patient continues with his rauwolfia. He begins to complain of stuffiness in the nose, dryness of the tongue, looseness of his stools; he may even comment on fullness of his face. His doctor reassures him that these are some of the annoying side effects. Then the patient off-handedly mentions that his sleep is becoming disturbed—he is beginning to have dreams, an occasional nightmare. He may even add that he is having fleeting waves of fear when he awakens or during the day. Regularly the significance of these symptoms is ignored by his doctor and he is given advice to continue with the tablets.

The chemically induced insanity is already on its way. If the doctor were only aware that these few off-hand remarks regarding dreams, nightmares and fears indicated that they had become very real to the patient! But the patient hesitates to discuss them.

Already the patient himself is aware of a change coming over his personality. The treacherous thing is that for several weeks, even months, these mental changes are only fleeting. A wave of pessimistic thoughts and ideas crosses his mind; he shakes his head, and it is gone. Then several waves of pessimistic and depressed thoughts assail him; he begins to wonder if he is going insane. The rest of the day he feels quite indifferent, perhaps "tranquilized", so he proceeds to talk himself out of the fear of insanity.

On a subsequent visit to the doctor he mentions that he gets "a bit depressed at times" and asks whether it could be due to the tablets. His doctor tells him that he may be a little depressed from them, but to persevere; after all, his blood pressure is down.

The florid picture of the rauwolfia reaction may develop rapidly, as early as three to four weeks after the patient begins to take the drug, or as late as after twelve months' continuous therapy.

Those who know this florid picture can diagnose it at sight. This rauwolfia reaction is a new clinical entity in psychiatry, to be distinguished from the well-known conditions of agitated depression and melancholia. Diagnosis is still a *sine qua non* of psychiatry.

The patients have a characteristic furtiveness; the roving of the eyes is a feature. They are peculiarly restless, their walk is robot-like, their thoughts are on a depressive and futile theme. Strangely the mood is not nearly so deeply depressed as the content of the ideas. There is a daily waxing and waning of the severity of the symptoms.

This is a deceptive state. A person has intensely wretched ideas, but unfortunately the mood and its accompanying facial behaviour are not so depressed as to give the idea of depression to the casual observer. This pattern develops in all types of persons, regardless of their pre-morbid personality.

A publican developed a florid reaction whilst taking rauwolfia. He became convinced that all the hotels would "go broke", that there was no future in the brewery business. There was great difficulty in undressing him when he was admitted to hospital. He was so convinced that he would lose all his money that eventually, when he was persuaded to strip, he had plastered onto his chest and abdominal wall £100 in £5 and £10 notes.

Three women thought that they were under the influence of the truth drug. Two felt compelled to tell about clandestine affairs they had been carrying on; the third, to tell her neighbours of some adolescent indiscretions.

These patients are often initially accused by their family of "putting on an act". However, their insane persistence in reiterating that they will lose their money etc., their undue restlessness, and the inability of family and friends to reassure them, place the condition in its serious perspective.

Incidentally I know of no condition in psychiatry in which an illness such as this can so exasperate and "get under the skin" of the non-afflicted partner. The husband's or wife's reactions to the patient are almost as diagnostic as the patient's. An explanation for this reaction has been given by a colleague who was treated with "Serpassil"; he developed mental indifference and sexual impotence.

The riddle of the nature of psychosis may lie hidden in the clinical paradox that rauwolfia relieves mental symptoms in psychotics, and induces insanity in normal people. Perhaps here is a clue to the chemical concept of psychoses.

What treatment is there? Unfortunately, there is no way of quickly reversing these mental symptoms. They may continue for months. The condition responds so poorly, if at all, to electroconvulsive therapy that it is doubtful whether it is worth while. Mainly the patients present a true problem of nursing management for the psychiatrist, the hospital staff and the relatives. Eventually, after a considerable duration, their insanity resolves.

Patients taking rauwolfia extracts should be carefully supervised, and an awareness of these mental complications kept to the fore. Some patients have been given 200 tablets, and a "repeat", before they are required to report back to

their doctor. Those practitioners who have themselves seen the disastrous effects of this drug have come to abandon its use.

Never before has there been such a challenge to doctors to look at the person as a whole, to see his symptoms against the background of his total personality. The indiscriminate use of tablets for the allaying of symptoms is not good medicine.

I am very sceptical of tranquillity's being obtained from a pill. Happiness comes as a consequence of pursuing a course of action. Some twenty-five years ago William Osler pointed out a way of life which leads to equanimity.

A BIOCHEMICAL APPROACH TO CALOMEL-INDUCED MERCURIALISM AND TO THE AETIOLOGY OF PINK DISEASE.

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In a previous paper (Barrett, 1957) the following hypotheses were developed: (i) that some of the symptoms ascribed to pink disease are predisposing conditions to mercurial poisoning from ingested calomel rather than early symptoms; (ii) that, in addition to calomel, another factor in the aetiology of pink disease is an unusually high alkalinity in the intestines.

These hypotheses were based on the following criteria: (a) increased absorption of mercury by laboratory animals given calomel under conditions simulating anorexia; (b) in-vitro experiments, in which calomel, suspended in a buffer solution, was oxidized immediately to the mercuric state at moderate alkalinities—small amounts at a pH of 8.4 and large amounts at a pH of 8.7; (c) the occasional occurrence of pH values as high as 8.75 in the stools of infants suffering from enteritis; (d) the differences in the early clinical picture of pink disease—for example, enteritis, constipation and achlorhydria; (e) the alkalinity of the intestines in such conditions; (f) the comparatively rare occurrence in the intestines of that alkalinity required for the rapid oxidation of calomel; (g) the finding of inflammatory processes in the ileum in enteritis associated with pink disease; (h) the toxicological approach to symptoms of mercurial poisoning observed in pink disease in Australia, instead of theories of "hypersensitivity" and "idiosyncrasy"; (i) the differences in the severity of the disease.

Acute mercurial poisoning from ingested calomel has now been produced in laboratory animals under the alkaline conditions stipulated in the hypothesis.

Calomel and Mercurial Poisoning.

Experimental Investigation.

A brief outline only of the experimental procedures and results is given here. The mercury determinations were carried out by the writer's method (Barrett, 1956).

Experiment I.—Young albino rats were given (a) daily oral doses of calomel and (b) equivalent doses of mercuric acetate, suspended in a vegetable oil. The dose corresponded, on a body-weight basis, to one well-known brand of teething powder containing calomel (Table I). Metabolism tests showed that approximately 20% of ingested mercury was absorbed in both groups (a) and (b). The growth curves corresponded to those of the controls. The animals were killed after having received the daily dose for ten to thirteen weeks. The mercury contents of the livers and kidneys in both groups (a) and (b) were respectively 0.1 milligramme and 6.6 to 7.7 milligrammes per 100 grammes of liver and kidney (wet tissue) (Table I). No significant difference in blood non-protein nitrogen content was observed between test and control animals.

Experiment II.—Young albino rats were fed (a) calomel and (b) mercuric acetate. These salts were incorporated in the diets at the following concentrations of mercury: 10, 50, 150 parts per million. The experiments were continued

for twelve months. The rats on the lowest dose grew normally. Growth was slightly retarded in both groups on 50 parts per million of mercury, but a marked decrease in growth was observed in the rats fed at a level of 150 parts per million, particularly in the calomel group. Restricted controls showed that the retardation of growth was due almost entirely to a diminished food intake, and except in a few cases only slightly to the direct effects of the drug. Two of the rats, after twelve months on calomel at the 150 parts per million level of mercury, and showing the greatest depression of growth, died unexpectedly. The mercury figures were 1.35 milligrammes per 100 grammes of liver and 15.5 milligrammes per 100 grammes of kidney; these approach Sollman's figures for acute poisoning (*vide infra*). In all cases the concentrations of mercury in the tissues of rats fed calomel at the 150 parts per million level were significantly higher than those of the corresponding mercuric acetate group (Table I). These figures agreed with those of the metabolism tests, at least 30% of ingested mercury from calomel and 20% to 25% of the mercury from mercuric acetate being absorbed. There was little or no difference in the mercury figures between groups (a) and (b) on the smaller doses. Blood analyses revealed slight but significant azotemia in the rats given 150 parts per million of mercury (both salts), compared with the controls.

This experiment demonstrated that the increased absorption of mercury from calomel was associated with loss of appetite. In this connexion, attention is drawn to the fact that anorexia or severe anorexia is a commonly ascribed early symptom of pink disease.

Experiment III.—Experiment I was repeated with calomel only, except that the calomel was suspended in a 60% dextrose solution and given to fasting rats. Equivalent doses, on a body-weight basis, were also given to half-grown rabbits, fitted with wooden collars to prevent refection. It was observed that the rabbits ate less under such conditions, and therefore simulated the anorexia observed in infants with pink disease. The ranges of pH of morning faeces were as follows: (i) normal rats, 7.2 to 7.6; (ii) fasting rats receiving calomel, 7.6 to 8.0; (iii) anorexic rabbits receiving calomel, 7.2 to 8.4. Intermittent diarrhoea was observed when the pH of the stools was 8.4. After several weeks on the daily calomel dose, nine rats, while in the fasting state, and five rabbits were killed. At the post-mortem examination the pH of the contents of the upper part of the duodenum was usually 8.0, compared with 7.2 to 7.6 for normal animals. The mercury contents were 0.67 and 17.9 milligrammes per 100 grammes of rabbit liver and kidney respectively. These figures agreed with the metabolism tests, 40% of ingested mercury being absorbed, compared with 20% to 30% in the previous experiments.

The daily calomel dose was continued with the remainder of the animals. One rat suddenly became ill and died. At the post-mortem examination the pH of the duodenum was 8.0 and that of the lower part of the bowel 8.5. The results of mercury determinations were 1.24 and 19.4 milligrammes per 100 grammes of liver and kidney respectively. One of the rabbits then suddenly became very ill. The pH of the stools increased from 8.0 to 8.7 or 8.8. Blood and mucus were found in the loose stools, but no pus. At the post-mortem examination the pH of the large intestine and of the terminal portion of the ileum was 8.7 to 8.8. The results of mercury determinations were 2.01 and 18.1 milligrammes per 100 grammes of liver and of kidney respectively. The liver and blood figures were of the same order as those in acute and fatal mercuric bichloride poisoning (Sollmann, 1948). *Proteus mirabilis* was isolated from the rabbits' faeces and also from several of the other test animals.

Attempts to reproduce the acute mercurialism by feeding the remainder of the test animals with *Pr. mirabilis* while in the fasting condition have so far failed. This work is being continued.

Discussion.

Sollmann (1948) states that, after clinical acute and fatal mercuric bichloride poisoning in man, the concentration of mercury in the kidneys averages 3.8 milligrammes and in the liver 1.9 to 2.6 milligrammes per 100 grammes of fresh tissue. Comparison of these figures with those of experiments I to III (Table I) shows (a) that the total amount of mercury accumulated in the kidney tissue is not necessarily an index of mercurialism, and (b) that the concentration of mercury in the liver is an index of the severity of mercurialism. A few blood mercury figures also support this, but, like urinary figures, are less reliable, owing to the rapid disappearance of mercury from the blood (Ashe *et alii*, 1953).

The symptoms of calomel-induced mercurialism observed in pink disease may be briefly explained as follows. Mercuric mercury reacts readily with sulphhydryl groups and so inhibits thiol enzymes (Fildes, 1940). The severity of the poisoning will be proportional to the maximum concentration of circulating mercuric ions, which in turn will depend primarily on the rate of absorption and secondarily on the rate of excretion. The rate of absorption of mercury from ingested calomel will be determined by the rate of oxidation to the mercuric state, which will be controlled by the pH of the intestines, as shown in the animal experiments.

The pH of the human pancreatic secretion ranges from 7.8 to 8.4, the pH being maintained at about 8.1 to 8.4 after a carbohydrate meal and decreasing to about 7.8 after a protein meal (Bodansky and Bodansky, 1952). The former pH values are highly suggestive, in view of the hypofunction of the parietal cells of infants aged under one year, and the associated difficulty of peptic digestion of the increased casein of cow's milk. From in-vitro experiments, calomel is oxidized to the mercuric state at a pH of 8.4—that is, at such a rate as to favour rapid absorption from the duodenum.

The acute mercurialism, which developed suddenly and spontaneously in a few of the test animals given the usual therapeutic dose of calomel while in a fasting or anorexic condition, was obtained when the pH of the ileum and large intestine was observed to increase from 8.0 to 8.7 or 8.8 as anticipated from the test-tube experiments. *Pr. mirabilis* was isolated from the stools of these animals.

In this connexion, Seviatt (1945) fed young rabbits with broth cultures of recently isolated strains of *Proteus*, but was unable to reproduce enteritis. The pH of the rabbits' faeces was not stated. The optimum pH for the production of deaminases by *Proteus*, with production of ammonia, is 8.0 (Anderson, 1946; Gale, 1940). *Pr. mirabilis* has a greater proteolytic activity than *Pr. vulgaris*, and is much more commonly found in human infections of the gastrointestinal tract (Wilson and Miles, 1955). The condition for proteolysis—*viz.*, a pH of 8.0 in the large intestine—was present in most of the calomel-treated animals; but it would appear that another factor was required for the development of high pH levels.

It can be calculated, from the experimental data, that some 40 milligrammes of calomel could be present in the lower part of the bowel of infants from a therapeutic dose, for immediate oxidation at pH values of 8.7 and higher, before expulsion of calomel from the loose bowels of infants with enteritis.

It is evident that a few calomel-containing teething powders, harmless as such, and given to millions of infants without any apparent effect, can cause, under certain unusual conditions, acute reactions to mercury. At present it is not possible to estimate the minimum lethal dose of "mild" mercurous chloride under such conditions (*vide supra*).

It is stressed, however, that calomel does not form a complex with proteins and, therefore, the poisoning is more insidious and less amenable to immediate treatment, and on theoretical grounds the minimum lethal dose would be less than that of mercuric compounds under the specified conditions (*viz.*, pH of 8.7 or higher in the ileum and large intestine—see also Table I).

Evidence has been produced, in accordance with the hypothesis, that calomel is (a) harmless as such, (b) relatively non-toxic when ingested under normal conditions, (c) chronically toxic when the pH of the duodenum is 8.0 or greater, (d) subacutely toxic when the pH of the duodenum approaches 8.4, (e) acutely toxic when the pH of the lower part of the bowel is 8.7 or higher.

As a matter of urgency, it is recommended that calomel be officially regarded as a poison.

The Aetiology of Pink Disease.

Age Incidence of Pink Disease.

Of Southby's 300 patients (Southby, 1948), 82% were aged between six and twelve months at the onset, "with

TABLE I.
Concentrations of Mercury Found in Tissues from Ingested (a) Calomel, (b) Mercuric Acetate, and (c) Mercuric Chloride.

Experiment Number.	Animals.	Daily Dose. (Milligrammes per Kilogram of Body Weight.)	Duration of Exposure.	pH of Intestines.		Deaths.	Concentration of Mercury in Tissue. (Milligrammes per 100 Grammes.)		
				Small.	Large.		Blood.	Liver.	Kidney.
I (a) ..	10 rats.	6	12 weeks.	7.2 to 7.6	—	NIL.	—	0.12	7.7
I (b) ..	6 rats.	8	6 weeks.	—	—	NIL.	—	0.08	6.6
II (a) ..	14 rats.	10	52 weeks.	7.2 to 7.8	—	NIL.	—	0.51	10.7
II (a) ..	2 rats.	10	{ 52 weeks. Unknown.	—	Probably 8.5	2	—	1.35	15.5
II (b) ..	16 rats.	14	52 weeks.	7.2 to 7.6	—	NIL.	—	0.22	9.1
Controls ..	16 rats.	0	52 weeks.	7.2 to 7.6	7.2 to 7.6	NIL.	—	0.003	0.03
III (a) ..	9 rats.	6	6 weeks.	7.6 to 8.0 ¹	7.6 to 7.8	NIL.	—	0.34	16.0
III (a) ..	1 rat.	6	{ 6 weeks. 4 days.	8.0	8.0 8.5	1	—	1.24	19.4
III (a) ..	5 rabbits.	6	6 weeks.	—	7.2 to 8.0	NIL.	0.031	0.67	17.9
III (a) ..	1 rabbit.	6	{ 6 weeks. 2 days.	8.0	8.0 8.7	1	0.067	2.01	18.1
Mercuric chloride (Sollman, 1939)	Rat.	50	24 hours.	—	—	All animals.	—	—	—

¹ In the human cases recorded by Sollmann (1948), in which the toxic substance was mercuric chloride, the patients died rapidly. The concentrations of mercury in the tissues were as follows (milligrammes per 100 grammes of tissue): blood, 0.015 to 0.12; liver, 1.9 to 2.6; kidney, 3.8.

an outstanding preference for the ninth month". The remainder were aged between thirteen and twenty-nine months at the onset. Of Cheek's patients (1950), 90% were aged from six months to two years. On the other hand, the majority of cases in Europe occurred in children aged over two years (Warkany and Hubbard, 1953).

Warkany and Hubbard (1953) ascribed the differences in the age distribution of children with acrodynia (pink disease), reported from the British Commonwealth and from Europe, to different "mercurial habits". In England and Australia the use of calomel teething powders was widespread, whereas in Europe antihelminthics containing calomel were given to older children. Although it would be impossible to obtain accurate figures for "mercurial habits" covering the period of Southby's cases, it would appear that, in general, calomel teething powders were given to Australian infants between the ages of six months and two years—that is, during the teething period.

Age Incidence of the Predisposing Conditions.

Achlorhydria.—In the absence of hydrochloric acid, the pH of the duodenal contents would be higher than that found in fasting animals (pH 8.0). Further, Southby (1949) observed that infants with achlorhydria had a history of diarrhoea, apparently non-infective, the motions being strongly alkaline, particularly during or after a meal. Nearly all the infants were aged under one year. Southby (1949) observed an accompanying or associated achlorhydria in very ill children suffering from pink disease.

Constipation with Alkaline Stools.—Collins (1948) stated that the increased amount of casein in cow's milk tended to produce constipation with alkaline stools. This observation is significant in view "of an outstanding preference for the ninth month" in the incidence of pink disease. Constipation favours retention of calomel in the bowels. Pharmacologists (for example, Sollmann, 1948) state that if calomel is retained in the intestines for any length of time, all the mercury may be absorbed, giving rise to systemic poisoning. They generally recommend a laxative some hours after the administration of calomel. Under increased alkaline conditions, as found in constipated infants, particularly during and after weaning, the poisoning would be more severe. Wood (1921) observed that constipation was frequent in his cases; in other cases the bowels were open regularly. The majority of his patients "were between 8 and 18 months".

Gastro-Intestinal Disturbances.—"Diarrhoea and enteritis under two years" is a universal condition, and various bacilli, including *Proteus*, dysentery bacilli and paracolon bacilli, have been regarded as the causative organisms (Sevitt, 1945). Evidence is produced in this paper that alkali begets alkali, and that under certain conditions—for example, proteolysis by *Pr. mirabilis*—calomel can produce acute mercurialism. Southby's cases of pink disease with inflammatory processes in the ileum (1948) may be explained on this basis. The isolation of *Pr. mirabilis* as another factor in pink disease explains the occasional outbreaks of pink disease observed by Southby (1948). Wood (1921) isolated *Bacillus dysenteriae* (Shiga) in one case of pink disease. In the stools of dysentery patients, Dods (1929) obtained a few pH values of 8.5.

Anorexia.—Anorexia may occur during the course of any physical illness in infants or during the introduction of solid foods in the "educational diet" (Collins, 1948). Marked anorexia is a common early symptom in pink disease. Previous illnesses or infections from which the baby "never seems to have recovered completely" is common in pink disease. The alkaline reaction of the intestines in anorexic conditions favours oxidation of calomel, as shown in the animal experiments.

Acrodynamic Symptoms.

The following hypotheses are proposed to explain the acrodynamic symptoms of pink disease: (a) that a vitamin B₆-containing enzyme is specially sensitive to mercurials at the active -SH centres of the protein component; (b) that the other factor is an alkali-induced deficiency of the essential fatty acids (linoleic and arachidonic acids); (c) that in non-mercurial cases of pink disease there is a double deficiency of vitamin B₆ and essential fatty acids.

It will be observed that this hypothesis parallels the findings of Peters *et alii* (1946) on arsenical poisoning—namely: that the pyruvate oxidase system was specially sensitive to arsenicals; that the attack was primarily upon -SH groups in this system; that the point of attack was on the protein component of the system; that there was a similarity between the clinical manifestations of arsenical neuritis and the neuritis accompanying vitamin B₆ deficiency.

Experiments are in progress to produce pink disease (mercury-induced acrodynia) in rats, in accordance with the foregoing hypothesis.

Hutchison (1935) cited Peters, who stated that a vitamin deficiency in pink disease was suggestive, but by no means certain.

The content of linoleic acid in breast milk is much greater than in cow's milk (Hilditch and Meara, 1944), and various workers have shown that the absorption of fat is reduced and variable in infants.

Hansen, in a series of papers (*vide* Sebrell and Harris, 1954), observed that the incidence of eczema and other skin conditions in infants was always associated with poor fat absorption, malnutrition and recurrent respiratory infections. He found lower iodine numbers in serum fatty acids of eczematous infants, and claimed that improvement followed the addition of fats rich in essential fatty acids. However, he cited other workers who reported no improvement.

Low plasma tocopherol levels have also been reported in diseases in which intestinal absorption is defective—for example, coeliac disease and diarrhoea associated with achlorhydria.

It is noted that respiratory infections were common in Hansen's patients, in his experimental animals with essential fatty acid deficiency, and in some of Southby's achlorhydric patients, and that bronchopneumonia is a common cause of death in pink disease. It is claimed that these disorders are manifestations of essential fatty acid deficiency, often alkali-induced.

It would appear, therefore, that intestinal alkali plays a dual role in the aetiology of pink disease (infantile acrodynia): (a) it renders mercury available to poison sulphhydryl enzymes; (b) it decreases the intestinal absorption of essential fatty acids and associated essential nutrients, already depleted by malnutrition and dietetic errors.

With regard to vitamin B₆ deficiency, (a) the essential fatty acids appear to have a sparing action on pyridoxine, and (b) alpha-tocopherol protects experimental animals against a restricted intake of vitamin B₆ and essential fatty acids (Sebrell and Harris, 1954).

The foregoing hypothesis offers an explanation for the following: (i) the acrodynic symptoms of pink disease; (ii) the reports of some cases of pink disease with no demonstrable association with ingestion of mercury—that is, a case of acrodynia due to a double deficiency of vitamin B₆ and essential fatty acid; (iii) the conflicting reports of dimercaprol therapy. It would appear that BAL is effective in atypical (mercurial, non-acrodynic) cases, partially effective in mercurial acrodynic cases, and ineffective in non-mercurial, acrodynic cases.

This theory is consistent with the age distribution of pink disease in Australia and England.

A rational treatment for pink disease, therefore, would appear to be the administration of (a) essential fatty acids, (b) vitamin supplements, (c) dilute hydrochloric acid and/or dimercaprol (BAL), depending on the clinical picture.

Classification of Symptoms of Pink Disease.

It would appear that the symptoms ascribed to pink disease may be classified as follows: (i) predisposing conditions, (ii) mercurialism and acrodynia, (iii) complications (superadded infections, prolonged undernourishment).

It was believed by some workers that calomel was given to infants after the onset of pink disease. One can understand their dilemma in the confusion of predisposing conditions and early symptoms, and the improbability of giving teething powders to healthy children.

Pink disease may be generally regarded as a deficiency disease induced by calomel and alkali, endogenous rather than exogenous, the main symptoms being those of mercurialism and acrodynia; but pink disease may also occur in the absence of mercurials.

Summary and Conclusions.

1. Acute and fatal mercurial poisoning has been obtained experimentally with oral therapeutic doses of "mild" mercurous chloride (calomel).

2. Another factor, *Pr. mirabilis*, appears to be involved in acute cases of calomel-induced mercurialism and of gastro-enteritis associated with pink disease.

3. A relationship between the age incidence of predisposing conditions, calomel teething powder habits and the age distribution of pink disease in Australia has been demonstrated.

4. It is postulated that: (a) a vitamin B₆-containing enzyme is specially sensitive to mercurials through the sulphhydryl groups of the protein component; (b) an alkali-induced, rather than a dietetic, deficiency of essential fatty acids accounts for the acrodynic symptoms of pink disease; (c) in non-mercurial cases of pink disease there is a double deficiency of vitamin B₆ and essential fatty acids.

5. The symptoms ascribed to pink disease have been classified as (a) predisposing conditions, (b) mercurialism and acrodynia, and (c) complications.

6. Pink disease may be generally regarded as a deficiency disease induced by calomel and alkali, endogenous rather than exogenous, the main symptoms being those of mercurialism and acrodynia; but pink disease may also occur in the absence of mercurials.

7. A rational treatment for pink disease has been suggested.

8. It is recommended, as a matter of urgency, that calomel be officially regarded as a poison.

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THE DIAGNOSIS OF ECTOPIC PREGNANCY.

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The diagnosis of ectopic pregnancy may present considerable difficulty. The clinical features of 500 cases of ectopic pregnancy treated at the Royal Women's Hospital, Melbourne, during the years 1950 to 1956, have been analysed in an attempt to assess their diagnostic significance.

Age and Parity.

The average age of the patients in this series was thirty-two years. This was considerably greater than that of women confined in this hospital—namely, twenty-six years. The majority of patients had had less than two previous pregnancies proceeding beyond fetal viability (Figure 1).

Past History.

There was evidence of previous pelvic infection at laparotomy in 29% of cases, although a history of this was elicited in only 9%. It therefore appears that pelvic inflammation predisposes to ectopic gestation.

The high incidence of previous abdominal surgery, 29%, is similar to that reported by Farrel and Scheffey (1943) and by MacFarlane and Sparling (1946). Previous appendicectomy had been performed in 80 cases (16%). The aetiological role of appendicitis in ectopic pregnancy cannot be assessed, because the incidence of the operation in a comparable series of the general population is unknown.

In 50 cases (10%) there was a history of previous ectopic pregnancy.

Symptoms.

The three most common symptoms were abdominal pain, amenorrhoea and abnormal bleeding *per vaginam*. The incidence of these and other symptoms is presented in Table I.

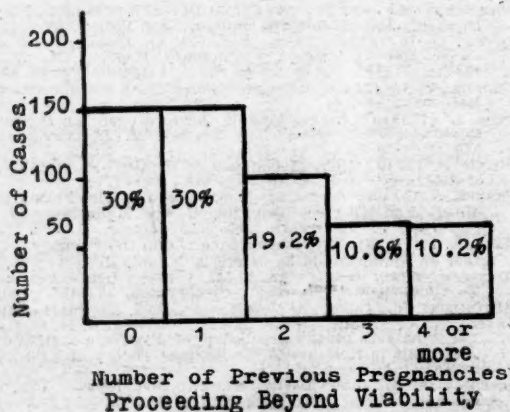


FIGURE 1.

Abdominal Pain.

Abdominal pain was present in all but one case. It was generally described as colicky or lancinating, but was sometimes dull and constant. The pain was usually situated in the hypogastrium. Less frequently it was generalized or in one or other iliac fossa. In a few cases it occurred in the epigastrium. It was referred to the shoulders in 39% of cases, and occurred on micturition in 14% and on defaecation in 11%.

Peel (1955) states that in most cases of ectopic pregnancy pain occurs before vaginal bleeding. This is con-

trary to the findings in the present series. Of the 430 cases in which there was a history of a relationship between these two symptoms, abnormal bleeding preceded pain in 280 (65%). The onset of the two symptoms was simultaneous in 77 (18%), and pain occurred prior to bleeding in only 73 (17%). Carabba and Silberblatt (1952) have reported a pattern of relationship similar to that of the present series. Pain is usually the patient's major complaint, and this may explain the belief that it frequently precedes abnormal hemorrhage *per vaginam*.

Amenorrhoea.

A period of amenorrhoea occurred in 375 cases (75%). The average duration was 7.7 weeks and the longest was 20 weeks.

Abnormal Bleeding per Vaginam.

Abnormal bleeding *per vaginam* occurred in 415 cases (85%) and was of three types. It followed a period of amenorrhoea in 278 cases (67%), it occurred in the intermenstrual phase following a normal period in 87 (21%), and there was abnormal loss at the time of menstruation in 50 (12%). In 350 cases it was possible to estimate the blood loss. This was stated to be less than that of a normal menstrual period in 203, heavier in 73 and equivalent to it in 130. The variation in both the type and amount of bleeding in this series indicates that it is the presence rather than the amount of hemorrhage which is of significance.

TABLE I.
An Analysis of the Symptoms in Cases of Ectopic Pregnancy.

Symptoms.	Number of Cases.
Abdominal pain	499 (99.8%)
Abnormal bleeding <i>per vaginam</i>	410 (82%)
Amenorrhoea	375 (75%)
Faintness	198 (40%)
Shoulder pain	190 (38%)
Vomiting	170 (34%)
Symptoms referable to pregnancy (morning sickness, breast changes)	115 (23%)
Diarrhoea	70 (14%)
Pain with micturition	70 (14%)
Pain with defaecation	55 (11%)

Faintness.

Faintness was present in 40% of cases. The incidence in other series varied between 25% (Crawford and Hutchinson, 1954) and 60% (Henderson and Bean, 1950). The variation probably results from differences, both subjective and objective, in the interpretation of the complaint.

Symptoms of Pregnancy.

Morning sickness occurred in 70 cases (14%), and breast changes in 45 (9%). There is little information concerning the frequency of occurrence of these symptoms, and it is recommended that future reports should include their incidence.

Gastro-Intestinal Symptoms.

The morning sickness of early pregnancy must be differentiated from the gastro-intestinal symptoms due to tubal rupture or abortion. Vomiting, which resulted from this rather than from the pregnancy *per se*, occurred in 170 cases (34%). Diarrhoea was present in 70 (14%).

Signs.

The most important signs were tenderness on abdominal palpation, pain on movement of the cervix and a tender mass or "bogginess" in one or other fornix (Table II).

Abdominal Signs.

Tenderness on abdominal palpation was found in 95% of cases. This was usually present in the lower part of the abdomen, often in one or other iliac fossa. Other, less frequent, signs included rigidity, rebound tenderness and distension. A mass was found in only 3% of patients, and in all cases this was due to a pelvic hematocoele. Cullen's sign was never observed.

Pelvic Signs.

Pain on movement of the cervix was present in 82% of cases. The pain radiated upwards from the pelvis and was usually severe. This test must be performed gently, and to elicit the sign it is unnecessary to move the cervix more than one to two centimetres.

TABLE II.

An Analysis of the Signs in Cases of Ectopic Pregnancy.

Signs.	Number of Cases.
General:	
Shock	124 (25%)
Temperature above 100° F. .. .	36 (17%)
Abdominal:	
Tenderness	470 (94%)
Rigidity	201 (40%)
Rebound tenderness	196 (39%)
Distension	70 (14%)
Mass	15 (3%)
Pelvic:	
Tenderness on cervical movement .. .	409 (82%)
Tender mass or "bogginess" in one fornix	360 (72%)
Cervix soft or blue	280 (56%)
Uterus enlarged	189 (38%)

The other important finding was a tender mass or "bogginess" in one fornix. This was present in one or other lateral fornix in 58%, and in the posterior fornix in 14%. In 95% of these cases, the detection and localization of this mass were confirmed at laparotomy. A soft or blue cervix was found in one-half of the cases. Uterine enlargement was detected less frequently. This may be due to the fact that pelvic tenderness often prevents accurate assessment of uterine size.

General Signs.

One hundred and twenty-five patients (25%) were admitted to hospital in a state of shock. In these patients the pulse rate was sometimes not raised. This may be due to the persistence of vagal inhibition as a result of pain.

In the present series, 86 patients (17%) had a temperature of 100° F. Crawford and Hutchinson have reported a similar incidence (15%). Therefore, the presence of a moderate degree of pyrexia does not exclude the diagnosis of ectopic pregnancy.

Clinical Features in Relation to Site and Outcome of Ectopic Pregnancy.

The exact diagnosis of an ectopic pregnancy is not usually possible until laparotomy. The site and outcome of the 493 tubal pregnancies are presented in Table III.

TABLE III.

Site of Tubal Pregnancy and Its Outcome.

Tubal Site.	Number of Cases.	Outcome when Observed.	
		Tubal Abortion.	Tubal Rupture.
Outer third	293 (56%)	144	128
Middle third	115 (24%)	48	59
Inner third	70 (14%)	4	65
Interstitial	25 (5%)	—	24
Total	493	196	276

A comparison of the clinical features of tubal rupture and abortion showed that the only significant difference was that shock occurred more commonly in tubal rupture (41%) than in tubal abortion (13%). The average period of amenorrhoea and the incidence of abnormal bleeding were almost identical.

There were 25 cases of interstitial tubal pregnancy. Eight of these patients were admitted to hospital in a state of severe shock. In one case there was the recognized association of a prolonged period of amenorrhoea with enlargement at the cornu of the uterus.

The three ovarian pregnancies and the one primary abdominal pregnancy had no specific diagnostic features. Two of the three patients with secondary abdominal pregnancies had periods of amenorrhoea lasting for twelve weeks. The third presented with a small bowel obstruction.

Special Investigations.

Certain procedures may be performed when the diagnosis is in doubt and the patient's condition is not acute (Table IV).

TABLE IV.

The Results of Special Investigations Used in the Diagnosis of Ectopic Pregnancy.

Investigation.	Number of Cases.
Findings on needling of pouch of Douglas:	
Blood	42
No blood	1
Result of pregnancy tests:	
Positive	40
Doubtful	30
Negative	16
Endometrial scrapings:	
Proliferative	16
Secretory	13
Decidual	34
White cell count (per cubic millimetre):	
Greater than 20,000	5
Between 10,000 and 20,000	23
Less than 10,000	23
Cervical smear findings:	
Infection	28
No infection	129

The pouch of Douglas was needled in 43 cases, and blood was obtained in 42. No complications arose from this procedure. Priddle and his associates (1952) found blood in 123 out of 129 cases of ectopic pregnancy. Failure to aspirate blood may be due to an organized haematocoele or an intact tubal pregnancy.

Pregnancy tests using the male toad and the immature female rat were performed in 86 cases. The results were positive in 40 cases, doubtful in 30 and negative in 16. If the result is positive, the test is of value, as an ectopic gestation has then only to be distinguished from the complications of an intrauterine pregnancy or chorion-carcinoma.

The microscopic examination of endometrial scrapings is of little aid in diagnosis. In the present series a decidual reaction was found in 34 out of 63 patients. Other writers have found a lower incidence (Crawford and Hutchinson, 1950, 27%; Romney, Hertig and Reid, 1950, 19%).

In this series the white cell count of peripheral blood varied from 5000 to 25,000 cells per cubic millimetre. It is therefore of no diagnostic value, and may be misleading when used to distinguish ectopic pregnancy from such conditions as acute pelvic inflammation or functional disorders.

Bacteriological examination of cervical smears revealed the presence of infection in 10% of the 157 cases in which it was carried out. This investigation is, therefore, of limited value in the differentiation of ectopic pregnancy from pelvic inflammation.

Diagnosis.

The diagnosis of ectopic pregnancy can be made in the majority of cases after a careful history and examination.

The clinical picture depends largely on the amount of bleeding into the peritoneal cavity. Bleeding was not severe in the majority of cases, and consequently the

symptoms and signs were localized in the pelvis and lower part of the abdomen. Abdominal pain, abnormal bleeding per vaginam and amenorrhoea were the three most important symptoms. Abnormal bleeding usually preceded the onset of pain. Symptoms characteristic of early pregnancy were sometimes present. The most reliable signs were pain on movement of the cervix and a tender mass or "bogginess" in one fornix.

When intraperitoneal bleeding was severe, the clinical features were shock, faintness, shoulder pain, abdominal rigidity and release tenderness. In these cases the diagnosis of ectopic pregnancy was made from the menstrual history and the pelvic signs.

It was impossible to make an exact diagnosis in a few cases. These patients should be kept under observation in hospital, and it is in this small group that needling of the pouch of Douglas may prove conclusive.

Differential Diagnosis.

When an ectopic pregnancy produces severe intraperitoneal haemorrhage, it may be simulated by a ruptured ovarian cyst, a ruptured spleen, a perforated appendix or a perforated peptic ulcer. When intraperitoneal bleeding is less severe, it may be confused with acute appendicitis, uterine abortion, acute pelvic inflammation, torsion of an ovarian cyst, urinary tract infection or psychosomatic disease.

Sixty-five patients in this series were discharged from hospital before the final diagnosis of ectopic pregnancy was made. They were later readmitted. The provisional diagnosis in each case on first admission is shown in Table V.

TABLE V.
Provisional Diagnosis on Patient's First Admission to Hospital.

Provisional Diagnosis.	Number of Cases.
Uterine abortion	28
Abdominal pain for investigation	12
Acute pelvic inflammation	10
Ectopic pregnancy	10
Functional menorrhagia	3
No diagnosis recorded	2

In reviewing the histories of these cases, it was considered that in 30 the symptoms and signs suggested the presence of an ectopic gestation.

Summary.

1. The clinical features of 500 cases of ectopic pregnancy treated at the Royal Women's Hospital, Melbourne, have been analysed.
2. The average age of the patients was significantly greater than that of women confined in this hospital.
3. A previous ectopic pregnancy had occurred in 10% of patients.
4. The three most important symptoms were abdominal pain, amenorrhoea and abnormal bleeding per vaginam.
5. Abnormal bleeding per vaginam was usually found to precede the onset of pain.
6. The most reliable signs were pain on movement of the cervix and a tender mass or "bogginess" in one fornix.
7. A clinical differentiation between tubal abortion and tubal rupture could not be made.
8. The special investigations of value were needling of the pouch of Douglas and the pregnancy test.
9. The white cell count and bacteriological examination of cervical smears may be misleading.
10. The majority of ectopic pregnancies can be diagnosed from a careful history and thorough examination.

Acknowledgements.

We are grateful to Professor Lance Townsend for his suggestions and advice in the writing of this paper. We wish to thank the members of the honorary medical staff

of the Royal Women's Hospital, Melbourne, for permission to examine the histories of the patients concerned who were under their care.

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OBSERVATIONS ON PROPHYLAXIS OF MEASLES WITH GAMMA GLOBULIN.

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SINCE the commoner complications of measles—bronchopneumonia and otitis media—usually respond well to antibiotics, at least one authority (McLorinan, 1953) questions whether sero-prophylaxis of contacts of measles patients is justified. However, the average child with morbilli suffers from loss of appetite and loss of weight for a week or longer and may become quite debilitated, and the persistent cough may seriously disturb the sleep both of the child and of his parents. The rare complication of measles encephalitis is said to carry a 10% or higher mortality rate (Fox *et alii*, 1953).

TABLE I.

Case Number.	Age of Child (Years and Months.)	Time of Injection. (Number of Days after Rash in Older Sibling.)	Gamma Globulin. (Cubic Centimetres.)	Result.
I	2 4	1	2	No measles.
II	1 2	1	2	No measles.
III	1 8	2	2	No measles.
IV	0 7	2	2	No measles.
V	0 7	2	2	No measles.
VI	2 6	2	1.6	Transient partial rash eight days later.
VII	1 6	2	2	Very mild attack eight days later.
VIII	3 0	2	2	One-day rash about two weeks later.
IX	2 3	2	1.75	No measles.
X	3 8	2	2	One-day rash.
XI	2 3	2	1.7	No measles.
XII	1 0	3	2	One-day rash one week later.
XIII	1 10	3	2	No rash; child irritable.
XIV	1 2	3	2	Three-day rash commencing four days later.
XV	0 2	3	1	No measles.
XVI	1 11	4	2	One-day rash.
XVII	2 2	6	2	Average attack of measles.

During an epidemic of measles in the latter half of 1955, I felt it desirable at least to ameliorate attacks of measles in the younger siblings of patients with this disease. This was accomplished by injecting two cubic centimetres or a little less of γ globulin intramuscularly within a few days of the appearance of a measles rash in the older sibling. The γ globulin was obtained from the Commonwealth Serum Laboratories. The syringe and needles used were sterilized by boiling in water, and the injection was given into the upper half of the buttock.

Table I shows the age of each child injected, the time of the injection (that is, the number of days after the appearance of the measles rash in the older sibling), the amount of γ globulin injected, and the result.

There were no reactions attributable to the injection.

It can be seen that of the two patients injected one day after the rash, neither developed measles; of the nine patients injected two days after the rash, four developed a very mild attack amounting to a one-day rash only; of the four patients injected three days after the rash, two developed measles, one of which was an attack of normal severity; the one patient injected four days after the appearance of the rash had a very mild attack; and another injected six days after the rash had an average attack apparently not modified by the injection.

Discussion.

The value of these results is limited by the relatively small series of cases reported. In interpreting them, it is assumed that a high percentage of the measles contacts would have developed measles of average severity without the protection provided. The results suggest that for the amelioration of measles in a young measles contact the protective injection of γ globulin (Commonwealth Serum Laboratories) should be given within two days of the appearance of the measles rash in the older sibling, though given up to four days after it may still provide a useful result. It is generally considered that the appearance of the rash corresponds to the third or fourth day of exposure of a sibling contact. It follows that the protective injection should be given within five or six days of exposure to measles, though delay for two more days may not be too late to mitigate an attack. If the injection is given within two days of the appearance of the rash in the older sibling, the amount injected should be two cubic centimetres for a child aged between three and four years (Cases VIII and X), and rather less than this amount for a younger child.

It is possible that, even in those cases in which the injection of γ globulin appears to prevent any sign of measles, some degree of immunity may develop. In theory, however, it seems desirable to aim at the development of a mild attack, including a recognizable rash.

Summary.

Reasons are given for attempting to mitigate the severity of measles in young sibling contacts of measles patients.

The series of cases reported suggests that this can be accomplished by an intramuscular injection of γ globulin (Commonwealth Serum Laboratories), preferably within two days of the appearance of the measles rash in the older sibling.

For a child aged three to four years the amount injected should be two cubic centimetres. For a younger child less than this should be given.

There were no reactions to the γ globulin.

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Reports of Cases.

THE TREATMENT OF SEVERE TETANUS.

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PRIOR to 1954, all really severe cases of tetanus were fatal. In 1954 Lassen *et alii*, Shackleton, and Honey *et alii* reported success in desperate cases, achieved by con-

trolling the tetanic spasms with muscle relaxants, while respiratory function was maintained by early tracheotomy and intermittent positive pressure respiration. Others have reported success with this regime; but as the disease is comparatively rare, a true evaluation of the method is difficult at this stage. This case is reported in order to elucidate the problem and at the same time to emphasize some points in management which we believe to be important.

Clinical Record.

The patient, a coal-miner, aged forty-two years, stood on a nail on September 17, 1956. He received no treatment at this time. He felt well until five days later (September 22), when he noticed some stiffness of his neck and difficulty in opening his mouth. This passed off during the afternoon, but he awoke during the night with an exacerbation of the above-mentioned symptoms. A provisional diagnosis of tetanus was made and 1500 units of antitetanus serum were administered.

He was transferred to Saint Vincent's Hospital the next day, arriving at noon. On examination he was found to be a large, well-built man, very restless and with pronounced trismus and neck stiffness. There was moderate rigidity of his limbs and abdominal muscles, but no respiratory difficulty. His temperature was 100° F. in the axilla. On the sole of the right foot there was a puncture wound which was surrounded by a tender erythematous area.

The diagnosis of severe tetanus was made, and conservative therapy was commenced with antitetanus serum (80,000 units given intravenously, 10,000 units intramuscularly and 10,000 units around the wound), paraldehyde (10 millilitres given intramuscularly) and penicillin. The wound was widely excised one hour after the administration of serum. Several minor spasms occurred shortly after his admission to hospital, and then at 6 p.m. on the same day he had his first major spasm.

In view of his rapid deterioration and the short incubation period (five days), we decided to adopt the principles of therapy outlined by Lassen.

An immediate high tracheostomy was performed under general anaesthesia, and a short cuffed Magill tube was placed in the trachea. A polythene catheter was inserted into the saphenous vein at the ankle and a nasal intragastric tube passed.

His treatment now consisted of constant, complete curarization and intermittent positive-pressure respiration. He was nursed in a separate room with a resident medical officer and nurse constantly in attendance.

The curare ("Tubarine") was given intermittently (15 milligrammes) as required via the tubing of an intravenous saline drip apparatus. The total quantity of "Tubarine" used was 3220 milligrammes. At first, a dose of 15 milligrammes was necessary every fifteen minutes, but this interval increased, and at the time of cessation it was from one to one and a half hours. Injections of 30 milligrammes of a depot form of curare ("Oily Tubarine") were also used early in the course, but had little if any effect on the intravenous dosage necessary.

Sodium phenobarbitone, given intramuscularly, was used for sedation, in a dosage of six grains every six hours throughout the period of curarization. At no time did the level of narcosis appear to be inadequate. The total dose of soluble phenobarbitone used was 180 grains. When curarization was suspended, the sodium phenobarbitone was replaced by a shorter-acting drug, paraldehyde.

The trachea and bronchi were aspirated every two hours with a sterile Tieman's catheter. Vigorous chest physiotherapy and passive limb movements were employed every four hours. The patient's posture was changed every two hours.

The amount of secretion at first removed was very large, but this gradually diminished. An X-ray film of the chest was taken every day. On the second day, collapse of the upper lobe of the right lung was detected, and partially cleared by posturing. Bronchopneumonia of the lower lobe of the right lung later developed and proved difficult to control owing to the presence of antibiotic-

resistant organisms. Bronchoscopic examination was performed once only, on the fifth day, to remove thick inspissated secretions.

A fortified milk mixture containing adequate vitamins and 2000 Calories (120 grammes of protein, 74 grammes of fat and 160 grammes of carbohydrate) in two litres was used for nutrition. Ninety millilitres of this were given every hour via the nasal intragastric tube. The tube was aspirated before each administration. It was found that a total of five litres of fluid (milk, plus the intravenously administered dextrose-saline solution) per day was necessary for adequate fluid balance.

Estimations were made each day of the hemoglobin value, hematocrit, white cell count and serum electrolyte contents. Moderate leucocytosis occurred from the chest infection, and in the early stages the electrolyte contents indicated respiratory acidosis. For the first three days of therapy with the respirator, arterial blood samples were taken, and estimations of pH, carbon dioxide tension, oxygen saturation and whole-blood buffer base concentration were made. The blood pressure and pulse rate were noted each hour. An indwelling vesical catheter was used throughout.

The curarization was first reversed on September 26, by the administration of 2.5 milligrammes of "Prostigmin" (after three days' therapy). A major spasm occurred shortly afterwards, and curarization was recommenced. This reversal was repeated on the fifth, on the seventh and finally on the tenth days. On each occasion spasms recurred, but it was apparent that they were becoming less severe.

Severe pyrexia (temperature rise up to 105° F.) from cellulitis of the buttock due to an anaerobic *Clostridium welchii* developed on the thirteenth day of treatment. The patient appeared moribund, with tachycardia and a falling blood pressure. The intramuscular injection of "Chloromycetin", the intravenous injection of hydrocortisone (100 milligrammes in six hours) and surface cooling were used with good effect. The cellulitis localized to a gas-containing abscess, which was later drained without incident.

In the immediate post-paralytic stage the patient had pronounced generalized muscular rigidity and weakness and a bilateral foot drop. His mental state was one of great apprehension, requiring careful and sympathetic management. Third cranial nerve palsy was also noted. All the foregoing signs gradually disappeared over a period of four weeks. He was discharged from hospital, cured, on November 21.

Discussion.

Relaxant.

Tubocurarine was used to produce muscle relaxation, as it is well tolerated, serious side effects are non-existent, and its action is readily reversed. When used in this fashion curare should be given in doses sufficient to produce complete rather than partial paralysis, and respiration should be fully controlled. We believe that anything less than this leads to considerable difficulties with ventilation from bouts of hypoxia, carbon-dioxide retention and bronchospasm. Therefore, any return of muscle tone indicated the need for further tubocurarine. To lengthen the interval between intravenous injections, a depot form of tubocurarine (oily tubocurarine) was given intramuscularly. In the dosage recommended by the makers, this preparation appeared to be of little value.

Sedation.

Lassen *et alii* (1956) and Wilson *et alii* (1956) have drawn attention to the occurrence of severe bone-marrow depression in patients subjected to prolonged nitrous-oxide anaesthesia. For this reason, an alternative form of sedation with soluble phenobarbitone was chosen for this patient. This proved satisfactory, as the patient remembered nothing of his harrowing experience and there was no bone-marrow depression. However, in retrospect, it was thought that the dosage was unduly high, and that the slow excretion of soluble phenobarbitone made the

weaning period more difficult. A shorter-acting barbiturate would be preferable in the future.

Ventilation.

The "AGA" pulmospirator provided an efficient mechanical means of intermittent positive-pressure respiration. Throughout, the patient was ventilated with air. This was considered important, as alterations in the patient's colour then served as a guide to the efficiency of the artificial respiration. Cyanosis developed rapidly when the patient was under-ventilated for any reason. Such a colour change would not occur with the patient breathing an oxygen-rich mixture unless gross under-ventilation existed. In this way, dangerous levels of respiratory acidosis could develop and go unrecognized until there was serious depression of the vital centres with cardio-vascular collapse.

As further aids to the maintenance of efficient ventilation, hourly observations of the blood pressure and pulse rate were recorded, and blood pH and plasma carbon dioxide estimations were carried out daily. By these means a mild respiratory acidosis was detected early and immediately treated by adjusting the respirator.

Respiratory Complications.

A tracheotomy *per se* increases bronchial secretions, which tend to become mucopurulent at first and later thick and sticky. They may pool in the bronchi and cause serious atelectasis unless quickly and persistently removed by coughing or tracheo-bronchial suction. We believe that the prevention of respiratory complications depends on vigorous physiotherapy, with frequent tracheo-bronchial toilet by the use of sterile suction catheters, two-hourly changes in posture, adequate hydration of the patient and humidification of the inspired air. Bronchoscopy is used only for patients with persistent pulmonary collapse which does not respond to the foregoing regime. Then it should be carried out under general anaesthesia, through the mouth and with the tracheostome occluded. This patient had to undergo bronchoscopy once for persistent collapse of the upper lobe of the right lung.

Summary.

A case of severe tetanus is presented. The patient was treated by Lassen's regime of complete muscular relaxation and intermittent positive-pressure respiration through a tracheostome, carried on over ten days.

Tubocurarine was the muscle relaxant used. The depot form of tubocurarine was disappointing as a means of providing prolonged relaxation.

Nitrous oxide anaesthesia was purposely avoided, and soluble phenobarbitone was substituted as a form of sedative. There was no bone-marrow depression.

The patient made a complete recovery.

Acknowledgements.

We wish to thank Dr. Richmond Jeremy for permission to publish this paper and for much helpful advice during the management of the patient; Dr. N. Newton for performing the tracheotomy; Commonwealth Industrial Gases, Proprietary, Limited, for the use of the "AGA" pulmospirator and spiropulso; and the nursing and resident medical staff of Saint Vincent's Hospital, who provided a twenty-four-hour "cover" throughout the acute stage of the disease.

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A FATAL CASE OF PARATHION POISONING.

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PARATHION is an organic phosphate insecticide readily available without restriction throughout Australia. It is a very good insecticide, being toxic to a great number of insects and mites that do damage to various fruit and vegetable crops. Unfortunately it is also extremely toxic to man, and although it is prominently labelled "Poison", it is not generally known how very dangerous the substance is. It is a very powerful anti-cholinesterase, and thus its manifestations resemble those produced by excessive stimulation of the parasympathetic system. These manifestations can be divided into two groups, as follows: (i) muscarine-like action, with anorexia, nausea, excessive sweating and salivation, constricted pupils with blurred vision, excessive bronchial secretions and pulmonary oedema; (ii) nicotine-like action, with muscular twitchings, first in the eyelids and face, later extending down the neck, arms and whole body. In severe cases there is weakness of the respiratory muscles and eventually respiratory paralysis.

This poison can be readily absorbed through the skin, the respiratory tract, the conjunctiva and the gastrointestinal tract, and therefore great care should be taken when it is being handled.

Protective clothing should be worn at all times, together with an efficient respirator or dust mask to prevent inhalation of the poison. If any is spilt on the skin, it should be immediately washed off with soap and water. Persons using parathion or other similar organic phosphates should wash hands, arms and face with soap and water before eating, drinking or smoking.

The only antidote is atropine, which inhibits the muscarine-like effects of the poison. Persons using these insecticides should always carry 1/100 grain tablets of atropine sulphate, to be taken at the first signs of poisoning. In extreme cases 1/30 grain of atropine sulphate may be given hourly until improvement occurs.

Clinical Record.

A man, aged twenty-three years, was an orchard-spraying contractor with considerable experience in using sprays of various types. He had used parathion once before without ill effects. He prepared the spraying solution by mixing a container of concentrated parathion with a quantity of water in the vat of his spraying machine. He was not wearing protective clothing or a mask. He then proceeded with the spraying operation, with two assistants handling the hoses. From time to time he rolled cigarettes and smoked them without washing his hands. At lunchtime he said he felt "a bit sick" and did not eat very much. His two assistants were unaffected. About mid-way through the afternoon he had a severe headache, felt ill and decided to go home. That evening he consulted his doctor, and although he had no signs of poisoning was admitted to hospital for observation. However, he developed no further symptoms or signs and was not given any specific treatment. The headache soon cleared up, and on the fourth day after exposure to the poison he was discharged from the hospital, feeling well.

The following day he felt well, and after eating a good midday meal he went out to the orchard to drain the parathion out of the spray machine. At about 3 p.m. he was seen at the machine; but nobody knows exactly what contact he had with the poison while emptying the vat. At about 4.15 p.m. he returned home with ice-creams, eating one, and went to the bathroom to wash. After a few minutes he called to his wife to help him, saying that he felt weak and could not see properly. Soon he fell to the floor and his breathing became very distressed. An ambulance then conveyed him to the surgery.

On examination of the patient in the ambulance at about 4.30 p.m. cyanosis of the face and extremities was noted and there was some frothy mucus around the mouth and nostrils; the pupils were constricted, and

muscular twitching of the eyelids, face and arms was present. The patient was not breathing, but there was a faint pulse at the wrist. He was taken immediately to hospital, but failed to respond to restorative treatment.

At the post-mortem examination on the following day the only significant findings were pronounced post-mortem staining of the body, considerable oedema and congestion of all the organs, particularly the lungs.

Summary.

I have here recorded a case of poisoning by parathion, an organic phosphate insecticide. The death of this young man, in the prime of life, was due to his ignorance of the deadly nature of the insecticide, and because of that ignorance his failure to observe simple precautions.

It is surely absurd that in these days of oppressive regulations covering all varieties of drugs *et cetera*, unskilled persons can buy and use extremely toxic chemicals such as parathion without restriction or supervision.

EMERGENCY CRANIOTOMY FOR MIDDLE MENINGEAL HÆMORRHAGE IN COUNTRY PRACTICE.

By W. H. COATES,
Melbourne.

THE operation of craniotomy for extradural hæmorrhage is performed commonly in large surgical centres. The object of reporting this case is to remind the general practitioner in country areas that he may be called upon to perform this life-saving procedure without complex instruments and skilled assistance. In these cases death results directly from cerebral compression, and the blood loss is of secondary consideration.

Clinical Record.

On the morning of July 17, 1953, a young Italian wood-cutter was admitted to the Edenhope Hospital with the history of having been struck on the head and neck by a falling tree. It appeared that he had lost consciousness initially for a few minutes, and now complained of pain and tenderness in the back of his head and neck.

On examination of the patient, the positive findings were those of considerable posterior cervical tenderness, blood in the posterior part of the naso-pharynx, and mild mental confusion. His pupils were equal and reacted to light, and the peripheral reflexes were normal. There were general signs of shock.

A provisional diagnosis was made of fracture of the base of the skull and possible injury to the cervical part of the spine. No adequate transport was available for the transfer of the patient to a base hospital 75 miles distant.

During the next two hours, photophobia, irritability and lowering of the level of consciousness became evident. No localizing signs appeared. Lumbar puncture was performed, and this revealed evenly blood-stained cerebro-spinal fluid under increased pressure. Within minutes the patient became deeply comatose, with stertorous breathing. The left pupil dilated and both plantar reflexes changed to the Babinski pattern. The systolic blood pressure rose to 180 millimetres of mercury, and the pulse rate fell from 70 to 35 per minute.

Acute cerebral compression with commencing medullary coning indicated the need for immediate surgery. At the bedside, without anaesthesia, a vertical incision was made through the left temporal fossa. The skull was trephined in the classical area with the aid of a mechanic's drill and a half-inch diameter counter-sinking bit. This opening was enlarged by splitting off a fragment of squama with a pair of bone forceps borrowed from the neo-natal ward. A large extradural clot was gently scooped out, a drain tube was inserted, and the wound was loosely closed in one layer. There was immediate improvement in the patient's general condition, and he was transported to the

base hospital in the back of a utility truck. Here, with adequate surgical facilities, the skull was reopened by Dr. S. C. Fitzpatrick, and the torn anterior branch of the middle meningeal artery found and ligated.

Blood transfusion and careful nursing finally resulted in complete recovery.

Summary.

1. A case is reported of extradural hæmorrhage, requiring surgical treatment in a small country hospital.

2. The value of simple craniotomy and drainage as an emergency measure is indicated.

Reviews.

Modern Clinical Psychiatry. By Arthur P. Noyes, M.D.: Fourth Edition; 1956. Philadelphia and London: W. B. Saunders Company. 9½" x 6", pp. 630. Price:

THE fourth edition of this book is a worthy successor to the previous editions. The new setting of the type and chapter headings has resulted in more space being available, without any obvious increase in the bulk of the book.

The range of subject has been chosen to cover nearly all aspects of psychiatry that may be of interest to the clinician, with the usual introductory chapters on mental mechanisms, symptoms, history taking and examination of the patient. At the end of each chapter is a useful bibliography. The New Standard Nomenclature as adopted by the American Psychiatry Association is mentioned; but the limited usefulness of any classification in psychiatry is emphasized. There has been a wider attempt to use the psycho-dynamic and genetic concepts in clinical psychiatry. Those mechanisms that are most usually accepted have been elaborated in such a way that no one would have difficulty in following the argument. The book is free from the extreme opinions and theatrical extravagances that the clinician finds so tedious and useless.

Particularly noteworthy are the chapters dealing with the development of personality, and the emphasis laid upon attitudes and anxieties growing out of disturbances in interpersonal relationships. A useful chapter, devoted to sociopathic personality disturbances, appears to place in better perspective those personality variants that are usually described under the heading of psychopathic personality. However, we cannot but notice the tendency to suggest a completeness and satisfactory understanding of many of the causes and mechanisms of psychiatric illness. Perhaps this is needed if one is to avoid becoming too diverse, or on the other hand, nihilistic. This tendency may lead the student to believe that ideas accepted at present are based upon a very considerable body of fact that amounts to a scientific theory, whereas it is probable that these ideas are little more than an opinion. The future of psychiatry would seem to depend upon the industry, enterprise and clinical curiosity that can be instilled into the student.

In spite of these criticisms, Dr. Noyes presents the great wealth of accepted psychiatric thought and opinions in a clear and fluent style that makes his writing interesting and easy to follow. "Modern Clinical Psychiatry" must be considered one of the most comprehensive and lucid expositions of psychiatric thought and practice of today. It covers the ground the psychiatrist knows well, together with the less frequent conditions with which he may be expected to have some familiarity. It appears particularly suitable for post-graduate study.

The Principles and Practice of Diathermy. By Bryan O. Scott, M.R.C.S., L.R.C.P., D.Phys.Med.; 1957. London: William Heinemann (Medical Books), Limited. 8½" x 5½", pp. 200, with 146 illustrations. Price: 25s.

THIS book has been produced by Dr. Bryan Scott as a compact text-book on short-wave diathermy for the use of physiotherapy students. It is perhaps a pity that the words "short wave" were not incorporated in the title.

As a student's book, it has the merit of being clearly set out and well illustrated, and it is right up to the minute. It includes descriptions of such recent problems as self-tuning machines, frequency control, and radio interference, and has an excellent chapter on the effect of metal in the field.

It is a pity that the high standard of some sections is not maintained throughout the book as a whole; theoretical

aspects are generally dealt with well, but on the practical side it falls short. Dosage—a difficult but important aspect—could be made clearer, and there is virtually no discussion of the clinical indications for treatment. In its present form, the chapter on electrosurgery would be better omitted. In the all-important chapter devoted to regional application, some techniques are well illustrated; but there are some curious omissions. Bifurcated leads are used to treat two ankles at one time; but there is no mention of the commonly used foot-to-foot and hand-to-hand techniques. The author has a peculiar preference for the use of a blind cable as a condenser electrode on many occasions when it would seem much simpler and safer to use a cuff electrode. Space is devoted to the heart and mediastinum, regions rarely ever treated; yet discussion of pelvic heating is quite inadequate.

This book is well worth reading by every physiotherapist, and of particular value to teachers; it contains information of value to everyone interested in the field. As a text-book it could easily be much better.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Practical Dermatology", by S. M. Peck, B.S., M.D., and L. L. Palitz, M.D., Ph.D.; 1956. New York: Landsberger Medical Books, Incorporated. Distributed solely by The Blakiston Division of the McGraw-Hill Book Company. 8" x 5½", pp. 382, with 122 illustrations. Price: \$7.00.

Deals with the commoner skin conditions for the benefit of the general practitioner.

"The Year Book of Orthopedics and Traumatic Surgery (1956-1957 Year Book Series)", edited by Edward L. Compere, M.D., F.A.C.S., F.I.C.S.; 1957. Chicago: The Year Book Publishers. 7½" x 5", pp. 336, with 208 illustrations. Price: \$6.75.

One of the Practical Medicine Series of Year Books.

"Perinatal Loss in Modern Obstetrics", by Robert E. L. Nesbitt, Jr., M.D.; 1957. Philadelphia: F. A. Davis Company. Sydney: Angus and Robertson, Limited. 9½" x 6½", pp. 452, with 108 illustrations. Price: £6 7s. 6d.

A survey of the causes of perinatal deaths from academic and practical aspects.

"The Work of WHO, 1956: Annual Report of the Director-General to the World Health Assembly and to the United Nations." Official Records of the World Health Organization. 11" x 8½", pp. 246, with illustrations. Price: 10s.

The title is self-explanatory.

"Practical Clinical Psychiatry", by J. R. Ewalt, M.D., E. A. Strecker, M.D., Sc.D., LL.D., and F. G. Ebaugh, M.D.; Eighth Edition; 1957. New York, Toronto, London: McGraw-Hill Book Company, Incorporated. 9" x 6", pp. 478. Price: \$8.00.

The first edition was published in 1925. The book has been extensively revised since the previous edition of 1947.

"The Medical Clinics of North America", edited by H. S. Lawrence, M.D.; New York number, with 25 contributors; May, 1957. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 9" x 6", pp. 285, with illustrations and tables. Price: Paper binding, £6 15s.; cloth binding, £8 2s. 6d. per annum.

Contains a symposium of 20 articles on medical emergencies.

"Clinical Electrocardiography: Interpretation on a Physiologic Basis", by M. Gargberg, M.D.; 1957. New York: Paul E. Hoeber, Incorporated. 10" x 7", pp. 335, with many illustrations. Price: \$12.75.

The author aims to provide a visual method for teaching electrocardiography based on a knowledge of physiological principles.

"The Infectiousness of Human Tuberculosis: An Epidemiological Investigation", by Gerh. Hertzberg; *Acta Tuberculosea Scandinavica*, Supplement XXXVIII, 1957. Copenhagen: Ejnar Munksgaard. 9½" x 6½", pp. 146.

The results of experience in the Division of Tuberculosis at the Oslo Department of Health.

The Medical Journal of Australia

SATURDAY, AUGUST 17, 1957.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

RECENT OBSERVATIONS UPON SARCOIDOSIS.

SARCOIDOSIS is a disease with what may be termed a wide appeal. It is common enough to enter into the differential diagnosis of a variety of conditions seen in general practice; its protean manifestations on the one hand, and its occasional localization to one, or predominantly one, system on the other, pose special problems for the general physician, the thoracic specialist, the dermatologist and the ophthalmologist. Its history alone is a fascinating study in the gradual evolution of a more or less complete clinical picture from Hutchinson's first description of some of the skin changes over three-quarters of a century ago. Besnier and Boeck added their descriptions before the turn of the century; the eye lesions were described by Heerfordt in 1909 and the bone changes by Jüdling a decade later. Schaumann's classical monograph, which gave the first satisfactory account of the disease as a whole, and which included the first reference to pulmonary involvement, ensured the addition of the author's name to the eponymous nomenclature formerly popular: for some reason Hutchinson's name was never included.

In recent years much interest has been shown in those cases associated with abnormality of renal function, and in the effect on calcium metabolism produced by the development of "hypersensitivity" to vitamin D. The pulmonary manifestations have also been the subject of special study. They are frequently not as benign as was once supposed; they sometimes progress to produce chronic fibrotic changes which have a crippling effect. J. G. Scadding,¹ whose series may perhaps be biased by the inclusion of a

disproportionate number of "problem cases", found that about half his patients with radiological evidence of parenchymal disease ultimately developed mild or severe dyspnoea; the prognosis seemed rather better if at some stage of the disease there had been evident enlargement of the hilar glands. Functional studies in these patients have shown a decrease in compliance and a fall in diffusing capacity, the latter being indicative of a loss of effective surface area for gas transfer, with or without thickening of the alveolar membrane (alveolo-capillary block syndrome).

The intrathoracic lesions of sarcoidosis are, of course, Scadding's special study, and it is his extensive experience of these and of pulmonary tuberculosis which has enabled him to adduce much sound evidence for the tuberculous aetiology of sarcoidosis in at least a proportion of cases. His views on this subject have at times been misrepresented, and it may be of interest to summarize them here. He has emphasized² that at present the definition of sarcoidosis must be primarily histological. Unlike the word "tuberculosis", which implies a specific cause as well as a recognizable clinical and pathological entity, "sarcoidosis" carries no aetiological implications, and it should not be limited by including a reference to "unknown aetiology" in the definition. The term sarcoidosis may thus logically be qualified by a word indicative of aetiology when this is known: he uses "tuberculous sarcoidosis" to describe certain cases, and perhaps it would not be unreasonable to suggest that "beryllium sarcoidosis" might be applicable to chronic berylliosis. Failure to appreciate the logical basis of the terminology has been a source of some confusion; Scadding has never claimed that all cases of sarcoidosis are tuberculous. On the other hand, he has produced incontrovertible evidence that the borderline between sarcoidosis and tuberculosis cannot be accurately defined. In 10% of his series of 142 cases tubercle bacilli have been isolated from the sputum at some stage, but, with the exception of three patients in whom florid tuberculosis supervened, the isolation of tubercle bacilli was unaccompanied by any change in the clinical or radiological picture or in the level of tuberculin sensitivity. By contrast, a patient mentioned by P. Kerby³ developed sarcoidosis after recovery from tuberculosis; another contrasting group is formed by a few patients with established tuberculosis whose Mantoux tests, even to 100 tuberculin units, yielded persistently negative responses.

These observations raise the question of the significance of the tuberculin insensitivity frequently found in patients with sarcoidosis. The presence or absence of skin sensitivity does not cloud the diagnostic issue if a histological definition of sarcoidosis is accepted—and it is difficult to justify any alternative. Over two-thirds of Scadding's patients showed no skin reactions to 100 tuberculin units, but occasional positive results are found to one and ten units. Various workers have shown that this tuberculin insensitivity is relative and not absolute; a positive result in those patients whose response is negative to 100 tuberculin units may be elicited by testing with depot tuberculin (James and Pepys⁴) or with tuberculin combined with cortisone given locally or systemically (D. A. Pike and

¹ *Tubercle*, 1956, 37: 371 (December).

² *Proc. Roy. Soc. Med.*, 1956, 49: 803 (October).

³ *Lancet*, 1956, 1: 602 (May 5).

⁴ *Proc. Roy. Soc. Med.*, 1956, 49: 799 (October).

J. G. Scadding,¹ K. M. Citron²). Indeed, as Citron stresses in a paper which includes original observations as well as an excellent review of the subject, relative tuberculin insensitivity is merely one manifestation of a depression of skin sensitivity to a variety of antigens evoking a delayed type response. Thus he suggests that skin testing with *Candida albicans* antigen may provide a useful alternative to the tuberculin test as a diagnostic aid, particularly in communities in which negative results from tuberculin tests are becoming increasingly common as the prevalence of tuberculosis declines. Citron found a negative reaction to *Candida* antigen in 60% of 30 subjects of sarcoidosis and in 10% of healthy and of tuberculous subjects. About 20% of the control series showed no reaction to 100 tuberculin units, as did 72% of the patients with sarcoidosis. The value of using both tests is shown by the fact that only 2% of the control groups failed to react to both tests, whereas no response to either test was found in nearly half the patients with sarcoidosis. Incidentally, Citron reviews the reason for the unreliability of the Kveim test. Less incidentally, it is necessary to remember that depression of the delayed types of response to an antigen occurs in other conditions, notably the reticuloses and particularly Hodgkin's disease.

All these observations indicate, if indeed the definition adopted did not do so, that the diagnosis of sarcoidosis rests on the safest possible foundations only when it is histological. Biopsy material is most commonly obtained from lymph nodes, liver, conjunctiva or skin, but occasionally difficulty arises in the interpretation of the appearances. It is not always possible to differentiate the lesions of sarcoid from those of granulomata of varied causation, and indeed from non-caseating tuberculosis such as may occur in lymph nodes some distance away from frankly caseating glands.

In this review of some current concepts concerning sarcoidosis it is appropriate to mention a clinical variant of the condition recently described by K. M. Citron and J. G. Scadding.³ They report three cases in which the diagnosis may be regarded as established (one is a good example of "tuberculous sarcoidosis", bacilli having been demonstrated in the sputum on three occasions, tuberculin sensitivity remaining absent and biopsy material being compatible with sarcoidosis). The clinical features in these patients were strikingly similar: dyspnoea out of proportion to the apparent parenchymal involvement in the chest radiograph, associated with persistent wheezing, worse on exertion, in which an element of stridor was recognizable on both inspiration and expiration. Bronchographically, multiple short stenoses were demonstrated, chiefly at the origin of segmental bronchi. Episodes of collapse and consolidation were sometimes temporarily associated with these partial obstructions. Treatment with cortisone and antituberculosis drugs produced some benefit, probably attributable to the effect of cortisone upon associated "bronchospasm". In a patient with identical clinical features observed by B. Gandevia,⁴ improvement with similar therapy was shown by serial tests of ventilatory capacity to be largely if not entirely due to the relief of

"spasm". However, Citron and Scadding consider that the stenoses in one patient showed bronchographic evidence of improvement after treatment. In two of the three patients (and also in the additional patient mentioned above) bronchial biopsies showed the changes of sarcoidosis. The authors' contention is that the sarcoid bronchial lesions, or perhaps the pressure effect of involved lymph nodes, are responsible for the stenoses; this receives indirect support from the work of V. V. Kalbian,⁵ who found bronchoscopic and histological evidence of bronchial involvement in more than half of eleven consecutive cases of sarcoidosis. Prior to these studies, bronchial lesions, although occasionally described in the past sixteen years, have received little attention.

Any discussion of treatment is unfortunately less stimulating than is consideration of the aetiology and natural history of this fascinating disorder. Treatment with anti-tuberculosis drugs seems unavailing, and their combination with cortisone or prednisone appears to meet with limited success. There are, however, some outstanding exceptions, and certainly the combination should be given an extended trial in those patients who, having failed to resolve their pulmonary lesions spontaneously, begin to develop shortness of breath. Prognosis in the early stages is difficult, and in the later stages depressing.

Current Comment.

PROGRESS IN RADIOBIOLOGY.

THE fourth International Conference on Radiobiology was held in Cambridge from August 14 to 17, 1955, being attended by delegates from twenty countries. The proceedings, including 73 papers, are published in a rather large volume with the title "Progress in Radiobiology".¹ The papers are all in fairly condensed and concentrated form, yet the presentations are most readable, a tribute to editorial policy. At the conclusion of each paper is a record of the discussion on the subject, including questions asked and answers given. This feature adds much interest for the reader, as many important points have been brought out and so are on permanent record.

The great bulk of the papers deal with academic radiobiological research projects, and there are only occasional crumbs for the clinician. The book is divided into eleven sections, which show the scope of the material covered; *inter alia* there are sections on the effect of radiation on metabolism and enzymes, radiation physics, radiation genetics, chemical protection, studies on spleen, bone marrow and blood, the radiobiology of bone and radiation carcinogenesis. Brief summaries of a few representative papers will demonstrate the scope of the conference offerings. Professor Dainton contributes an important article on radiation chemistry in which he deals with oxidation and reduction phenomena, and the effects of radiation on large molecules. Wolff and Luippold discuss aspects of chromosome breakages induced by radiation, which are single-hit phenomena. Broken ends may heal and reconstitute the original configuration. When joins with broken ends of another chromosome are seen, the result indicates that two hits have been effective on two chromosomes. Studies have been made on the time required for healing, and on the protein synthesis which permits healing. Eldjarn and Pihl study the mechanism

¹ Brit. M. J., 1955, 2: 1126 (November 22).

² Tubercle, 1957, 38: 33 (February).

³ Thorax, 1957, 12: 10 (March).

⁴ Personal communication.

⁵ Thorax, 1957, 12: 18 (March).

⁶ "Progress in Radiobiology: Proceedings of the Fourth International Conference on Radiobiology Held in Cambridge on 14th to 17th August, 1955", edited by J. S. Mitchell, B. E. Holmes and C. L. Smith: 1956. Edinburgh and London: Oliver and Boyd. 9½" x 6", pp. 528, with illustrations. Price: 63s.

of chemical protective agents against ionizing radiation. The sulphur-containing protective agents cystamine and cysteamine have been studied, labelled sulphur being used in the compounds. These substances inactivate the products of irradiation of water by virtue of the reactivity of their sulphur groupings, and so fewer free radicals are available within an irradiated cell to produce evidence of radiation damage. Therkelsen shows that cysteamine administered to mice protects them from an otherwise lethal dose of nitrogen mustard. Pirie describes experiments on the production of radiation cataract. He reports that if the beam of radiation included the whole lens, uniform opacity resulted; if only the central portion of the lens received the same dose, no opacity resulted; if a marginal segment of the lens received the same dose, a marginal opacity resulted spreading segmentally to the centre. The conclusion is reached that irradiation of the peripheral cells produces some influence on the central cells which does not act in reverse fashion. Elson records the effects of a mustard, CB-1348, and the synthetic chemical "Myleran" in blood and bone marrow studies, and compares their action with radiation. Radiotherapy destroys the activity of all varieties of haemopoietic cells. "Myleran" reproduces only the myeloid damage, and CB-1348 produces lymphoid destruction. If both are simultaneously administered, a marrow and systemic response akin to that of radiation is seen. Belcher reports studies on the whole body irradiation of rats, and similar experiments with one limb shielded. Regeneration of irradiated marrow was much quicker in the animal with partial shielding, and it is concluded that a regenerative factor is produced by unirradiated haemopoietic tissue which stimulates the irradiated marrow. In several contributions the effects of the administration of Sr^{90} on bone growth and metabolism are reported; profound depression of both growth and metabolism is recorded. Mole writes on the carcinogenic effects of irradiation. Firstly a mutation may be produced in a cell, giving it neoplastic properties. Secondly, the radiation damage to the tissue may be repairable. In this repair process the neoplasm may arise, particularly if the radiation is long-continued and interferes with the subsequent repair. This is the hypothesis of "unstable repair" and is illustrated clinically, e.g., when carcinoma of the colon appears as a sequela of ulcerative colitis. Smithers, in the discussion of Mole's paper, rightly stressed the very low occurrence of neoplasms as a result of standard therapeutic irradiation procedures.

Bray and Glucksmann report studies of tumour production, using both radiation and chemical carcinogens. A non-carcinogenic dose of beta rays and a non-carcinogenic dose of chemical become synergistic and carcinogenic. The incidence of carcinomas is reduced and that of sarcomas is increased if a chemical carcinogen is applied to a region treated previously with a non-carcinogenic dose of beta rays, as compared with the incidence of those tumours when a carcinogen alone is applied. The explanation is that the susceptible epithelial cells have been destroyed by the beta radiation, while the beta rays produce an unstable scar in which the regenerating tissue is predisposed to tumour formation.

This book of proceedings should be perused by all those interested in radiation therapy; it is a necessity for the research worker interested in radiobiology. The volume is a credit to the editors, publishers and printers, and is a worthy record of a British-organized international conference.

LUPUS NEPHRITIS.

It is often stated that the characteristic lesions of the renal glomerulus in systemic *lupus erythematosus* are focal thickenings of the basement membranes of the capillary loops. The rigid appearance of these thickened loops is reminiscent of bent fencing wire, and they have come to be known as "wire loop" lesions. This form of membranous glomerulonephritis is fairly common in *lupus nephritis*, but according to R. C. Muerhcke, R. M. Clark,

C. L. Pirani and V. E. Pollak¹ it is not diagnostic unless accompanied by other more specific types of glomerular lesions, such as focal necrosis, hyaline thrombi and the presence of haematoxylin bodies. These authors investigated 33 patients suffering from systemic *lupus erythematosus* by means of renal biopsy. In 21 patients two or more specimens were studied by biopsy or autopsy at intervals varying between six weeks and sixteen months. From these studies the patterns of disease in the kidney became fairly clear. At first there is an irregular thickening of the basement membrane in the periphery of the glomerular tufts often associated with local proliferation of endothelial cells. This focal glomerulitis develops so that in many cases there results a conglutination of endothelial nuclei with narrowing of capillary loops until a local necrotic focus develops. In others typical membranous glomerulonephritis (wire loops) develops either alone or in combination with necroses. Fibroepithelial crescents, adhesions and gradual obliteration of glomeruli are found at the sites of necroses, and the appearance is then that of subacute glomerulonephritis. Evolution to chronic glomerulonephritis with granular contracted kidneys occurred in only three patients.

It appears, then, that the glomerulonephritis of systemic *lupus erythematosus* may be predominantly membranous, corresponding to Ellis type II nephritis, or it may be proliferative and resemble Ellis type I. Nine of the 33 patients had no clinical or histological evidence of renal involvement. Mild *lupus nephritis* was present in ten patients, in three of whom there was only histological evidence without abnormalities in the urine. The remaining patients had *lupus nephritis* of greater severity, and in some cases this was the dominant clinical feature of the disease. In four of these patients oedema was the presenting symptom, but in the rest renal involvement appeared at varying periods up to ten years or more after the first signs of systemic *lupus erythematosus*. There were seven patients who exhibited all the criteria of the nephrotic syndrome at some stage in the course of the disease. In addition there was a group of three patients with rapidly fatal *lupus nephritis* differing from the nephrotic syndrome only in being associated with normal blood cholesterol values.

In general, the renal manifestation of *lupus erythematosus* simulated ordinary glomerulonephritis chiefly in its subacute and nephrotic forms. It differed in having exacerbations at shorter intervals and by the invariable progression of the glomerular lesions despite any form of therapy. It is rare for *lupus nephritis* to be unaccompanied by other manifestations of systemic *lupus erythematosus*. Yet one must always consider systemic *lupus erythematosus* in the differential diagnosis of nephritis; and if there is any doubt, the histological evidence of the renal biopsy should solve the problem, even if the L.E. cell cannot be demonstrated.

TREATMENT OF INFANTILE HYDROCEPHALUS.

In a review of the current treatment of internal hydrocephalus in infants, Donald D. Matson² has emphasized that the first responsibility of the surgeon is to make an accurate assessment of the state of the hydrocephalus. The first decision must relate to whether the hydrocephalus is progressive or not. If it is, and if irreversible brain damage has not yet occurred, then the best possible operative treatment should be carried out at the earliest moment. The second possibility is that irreversible damage to the brain has already occurred to a degree that prevents an acceptable result, no matter how successful operative treatment may be; in such cases operation is futile and unwise. The third possibility is that, although hydrocephalus is present, there is no clear evidence that it is actively progressive at the time of the examination. Under these circumstances a clearly defined programme of repeated regular examinations must be carried out.

¹ *Medicine*, 1957, 36:1 (February).

² *New England J. Med.*, November 15, 1956.

Progression of hydrocephalus is most accurately assessed by regular estimation of intracranial pressure and by regular measurement of the head circumference. It should be remembered that if an infant's head is already enlarged it will not grow at all for a while if intracranial pressure becomes normal. It will wait until the body catches up. If the enlarged head continues to grow, therefore, even slowly, it means that there is still increased intracranial pressure.

The successful operative procedures are the various "shunt" operations designed to give continuous relief to the increased pressure of cerebro-spinal fluid. Operations aimed at decreasing the formation of the fluid have been uniformly unsuccessful. A number of shunt procedures have been used by Matson in a series of 210 patients over the last eight years. The first is a shunt by means of a polythene tube from the lumbar spinal subarachnoid space into the ureter after the removal of a kidney. This has been performed on 108 patients and has given by far the most satisfactory long-term results. Mechanical obstruction of the tube has been rare. Bad results have sometimes occurred from the acute dehydration of the infant due to fluid and electrolyte losses through the shunt, or from secondary infection spreading from the urinary tract to the meninges. However, between 60% and 70% of the children who have had this procedure are alive, symptomless and developing well after a follow-up period that ranges from a few months up to seven years. The second type of shunt is by means of a long subcutaneous tube from the ventricle to the ureter. This has been used only in patients with obstructive hydrocephalus. It is unsatisfactory from the long-term point of view because of the necessity to lengthen the tube with the growth of the child, usually every 12 to 18 months. This operation is no longer performed if other procedures can be made effective. The third type of operation is a peritoneal shunt by means of a plastic or rubber catheter from the lumbar subarachnoid space or from the lateral ventricle into the peritoneal cavity. This has been performed 155 times in 64 patients. The large number of operations in proportion to the number of patients reflects the difficulties encountered in making these shunts work satisfactorily. The problem of dehydration from fluid lost into the urinary tract is averted and the risk of infection decreased by this procedure, but nevertheless Matson states that it is not his first choice. The shunt from the ventricle to the cervical subarachnoid space for obstructive hydrocephalus has been carried out 38 times in 31 patients. It usually simply converts an obstructive hydrocephalus into a communicating one, and needs to be followed by a lumbar arachnoid ureteral shunt a week or ten days later.

Matson holds that the most fruitful lines of future development lie in two directions. The first is in improvement of the technical aspects of these shunt operations into the peritoneal cavity, so that a higher percentage of the shunts will function satisfactorily. The second is in the effort to develop some kind of artificial valve that can be inserted into these shunt tubes to permit absolutely competent unidirectional flow of fluid directly from the lateral ventricles of the brain into the circulating bloodstream, no matter what the pressures in the ventricle or venous system may be.

POLIOMYELITIS VACCINE.

A WORLD HEALTH ORGANIZATION report dated July 19, 1957, states that the World Health Organization Committee on Poliomyelitis, which was then meeting in Geneva, under the chairmanship of Sir Macfarlane Burnet, has come out strongly in favour of large-scale trials of a new poliomyelitis vaccine prepared from a living virus which has been "attenuated", so that it is no longer capable of causing the disease, although it may be expected to provide lasting protection against it. The live-virus vaccine can be given orally, instead of being injected. The experts are satisfied that preliminary trials of this vaccine carried

out by several different research teams have failed to reveal any signs of illness or other harmful effects either in the persons vaccinated or in members of their families. They consider therefore that the vaccine may now safely be given larger scale trials among the population.

At the same time, the World Health Organization experts do not suggest that this vaccine should displace the Salk type of killed-virus vaccine in those countries where it is now being used or is about to be used. They believe rather that the new vaccine should be an adjunct to the present vaccine, though it might eventually replace it if that is found desirable, or be a substitute for it where the use of the Salk vaccine is not feasible.

If the proposed trials of the live-virus vaccine prove successful, it may be hoped, according to the World Health Organization Committee, that the immunity it provides will be reliable and long-lasting, and may result in the elimination, or at least in a substantial reduction of the virulent strains of poliomyelitis virus at present in circulation. In the experts' opinion, the present killed-virus vaccine is not able to achieve this kind of result. The Committee emphasizes, however, that the most stringent precautions should surround the proposed trials of the new vaccine. In order to prove its complete harmlessness it should first be given to limited numbers of selected groups of persons. Furthermore, the properties of the attenuated strains of virus used in the vaccine should be measured in a number of different laboratories. It is recommended also that the trials be carried out on a voluntary basis and with the approval of the local authorities concerned. The Committee stresses that the object of the proposed trials of live-virus vaccine and the need for carrying them out are based on the belief that they may go far in further reducing the prevalence of poliomyelitis in the world.

The Committee has also reviewed the experience gained in those countries where killed-virus vaccine of the Salk type has been used in recent years. In France, poliomyelitis vaccination was not officially started before June 1, 1956. Several million doses have been released so far, but, since the majority were exported, a close follow-up of results will be possible only for about 800,000 vaccinations. Of these, 100,000 vaccinations had been given up to July 1, 1957, with negligible side reactions. In Poland, the average mortality from poliomyelitis in the years 1951-1954 was 6.3% of reported cases. More males than females caught the disease (55% against 45%), and the highest incidence rate was in the group nil to four years of age (78% of all cases). In 1957 Poland began the production of poliomyelitis vaccine; and in October next it is planned to vaccinate 100,000 children in Warsaw and Lodz. In Great Britain, vaccination has not offered complete protection against poliomyelitis, as some cases of paralytic disease were observed in vaccinated children. After the incidents which occurred during the first large-scale vaccinations in the United States of America, the authorities decided to manufacture a modified form of the Salk vaccine, the type 1 Mahoney strain of virus being replaced by a less virulent one. In Canada, the lower incidence rate of poliomyelitis during the last two years has made it difficult accurately to evaluate the results of the vaccination programme. In 1956, eleven cases of paralytic poliomyelitis were reported among a total of 1,860,000 vaccinated children, and 136 cases in 2,140,000 unvaccinated children of similar age. In South-East Asia, the poliomyelitis incidence has remained low. Cases among the adult population are almost unknown except among foreigners. In the United States of America, 15,400 cases of poliomyelitis occurred in 1956. This represents a rate of 9.2 per 100,000 population and is the lowest recorded since 1947. Because of the wide annual variations in incidence, it has not been possible definitely to attribute the low incidence in 1956 to the widespread use of poliomyelitis vaccine (70,000,000 persons were vaccinated). In Israel, all children from six months to three and a half years of age were vaccinated between January and May, 1957. This was done because it was found that over 80% of paralytic cases occurred in that age group. Among emigrants from Western Europe and the Americas up to

the age of forty years it was possible to vaccinate 130,000 persons. In the Union of South Africa, vaccination campaigns began in 1955 and were continued through 1956 and 1957. During this period the country experienced a prolonged and severe epidemic which, for the first time, affected Africans as much as people of European descent. However, among the Africans the disease affected young children for the most part; while among the Europeans older children were also affected, and there were many cases among adults. Up to the present about 500,000 children have been vaccinated. In the Union of Socialist Soviet Republics, there was a distinct rise in poliomyelitis incidence in 1955 (eight cases per 100,000 population). Available information indicated no reduction in 1956 or 1957. Young children under seven years of age were most affected (85% of cases). During the spring of this year 80,000 children were vaccinated. In Sweden, large-scale vaccination campaigns have begun in February, 1957, and some 800,000 persons have been vaccinated. No cases of poliomyelitis following inoculation have been reported.

SHOCK IN OBSTETRICS.

ATTENTION has been drawn to an important subject by Duncan E. Reid,¹ who discusses the various forms of shock encountered in the obstetric patient and the factors which contribute to it, and expresses his own views on prevention and therapy. Shock is described as a syndrome characterized by a decrease in effective blood volume associated with an insufficient cardiac output leading to peripheral circulatory failure. Conventionally shock is assigned to trauma with blood loss, to sepsis or to myocardial infarction. Shock in obstetrics is similar to that seen in medical or surgical conditions, and the treatment of the syndrome is identical. However, the term "obstetric shock", Reid points out, has been introduced to designate the circulatory change associated with amniotic fluid embolism or with the deposition of fibrin in *abruptio placentae*, supposedly from massive intravascular coagulation. Clinically, a fall in arterial pressure below 100 millimetres of mercury is a forewarning of impending shock, and the blood volume has already been reduced by some 30% (approximately two litres of blood). In shock due to sepsis the blood volume is not reduced except in cases of peritonitis. In shock with reduction of circulating blood volume vasoconstriction occurs, especially in the skin and kidneys. Oliguria and anuria often follow and persist until the arterial pressure is restored.

The types and causes of shock in obstetrics are discussed by Reid from a study of 306 maternal deaths from the Commonwealth of Massachusetts, 84 of which were due to shock. Deaths from shock comprised about 25% of the total maternal deaths. Uterine atony accounted for 41 deaths, nearly half the total number of deaths due to shock. There were eight deaths directly due to trauma of the birth canal, five from ectopic pregnancy, eight from accidental haemorrhage and *placenta praevia*, three from impairment of blood coagulation, six from acute inversion of the uterus, two from retained *placenta accreta* and seven from shock due to sepsis; one patient died of myocardial infarction, and two deaths were attributed to miscellaneous causes. The series also included seven deaths from amniotic fluid embolism and nine from pulmonary embolism. During a comparable seven-year period, 47 patients were treated at the Peter Bent Brigham Hospital on account of acute renal failure complicating shock in obstetrics. This series comprised 15 cases of post-partum haemorrhage, 11 of accidental haemorrhage, six of abortion, four of shock following transfusion reaction and four of *placenta praevia*. Thirteen of these 47 patients died, and it is noted that injury and blood loss accounted for nearly all the deaths from shock.

Reid briefly discusses these listed causes and makes certain comments on treatment. Timely packing of the uterus with gauze is advised in cases of atonic post-

partum haemorrhage not responding to routine treatment, and is preferred to bimanual compression of the uterus. Ill-advised mid-forceps extractions and internal podalic version are described as being responsible for deaths from traumatic shock in the series. Reid considers that extra-peritoneal Caesarean section still has a place in the treatment of the potentially infected patient. He stresses the importance of blood replacement in ante-partum haemorrhage, since a number of patients classified under "post-partum haemorrhage" lost their lives because of pre-delivery blood loss. The sudden appearance of severe respiratory distress during an otherwise normal labour is stated to be pathognomonic of amniotic fluid embolism. Attention is drawn to necrosis of the anterior lobe of the pituitary gland and acute renal failure as sequelae of obstetric shock. Reid stresses the importance of treating shock in obstetrics by restoration of the effective blood volume by rapid and efficient transfusion of whole blood. He concludes that deaths from shock in obstetrics are largely preventable, and that the condition cannot be treated effectively by blood obtained from a blood repository outside the hospital or from donor lists. His suggestion that no hospital be allowed to care for pregnant women if it is unable to meet this essential requirement is rather too drastic to be practicable, but it provokes some salutary thought.

RADICAL NECK DISSECTION FOR PAPILLARY CARCINOMA OF THE THYROID.

THE conventional block dissection of the neck when applied to papillary cancer of the thyroid is a parody of the principles of cancer surgery, has not increased the rate of cure and should be abandoned, according to George Crile, junior,¹ who needs no introduction in the realm of thyroid surgery. Crile points out that the jugular chain of lymph nodes is not a primary but a secondary zone of metastasis from most papillary carcinomas of the thyroid. He states that the most important primary zones of metastasis from papillary cancers are the centrally located lymph nodes of the superior mediastinum and the paratracheal nodes that lie behind the thyroid and along the trachea. Patients with papillary cancer of the thyroid do not die from uncontrolled lateral cervical metastases, but from invasion of the trachea, oesophagus and mediastinum by the primary tumour and its centrally located metastases. The lateral cervical metastases from thyroid cancers lie deep to the carotid, as well as superficial to it, and cannot be resected *en bloc* without sacrifice of the carotid vessels. In the vital central zone an *en-bloc* operation is not feasible, and the primary tumour and its central metastases must be treated by dissecting them free of the vital structures. Since there is no *en-bloc* operation which can encompass the primary cancer, its central and mediastinal metastases and its lateral cervical metastases, sacrifice of the sterno-cleido-mastoid muscle is a mere parody of the *en-bloc* principle.

The superior results from carefully planned conservative operations can best be explained, Crile suggests, by assuming that the standard neck dissections have neglected the vital central zone of mediastinal and paratracheal metastases. He states that many papillary carcinomas of the thyroid are, like endometriosis, amenable to hormonal control. Suppression of thyroid-stimulating hormone by daily administration of three or four grains of desiccated thyroid often results in regression of the tumour and its metastases. An important practical point is raised by Crile when he states that most of the patients with papillary carcinoma of the thyroid are teen-age girls or young women, in whom a block dissection is a tragic mutilation. He points out that since papillary carcinoma of the thyroid is reported to be better controlled as a result of well-planned excision of the primary tumour and the groups of involved nodes, the burden of proof lies on the surgeon who inflicts unnecessary deformity.

¹ *Am. J. Obst. & Gynec.*, 1957, 73: 697 (April).

¹ *Ann. Surg.*, 1957, 145: 317 (March).

Abstracts from Medical Literature.

PÆDIATRICS.

Obstruction of the Colon from Meconium or Faeces in the New-born.

J. L. EMERY (*Arch. Dis. Childhood*, February, 1957) discusses the abnormalities in the meconium of the fetus and new-born. He describes ano-rectal plugs of meconium and meconium plugs found elsewhere in the intestine, sometimes in the small, sometimes in the large intestine, and causing obstruction. The obstruction appears to be due to a segment of firm, tenacious meconium forming the tip of a cone at the obstructing point. In these children pancreatic function seemed to be normal. He describes meconium bodies—small, light yellow particles, which are sometimes found in the meconium of the new-born. While these are usually very small, they are occasionally large, and produce obvious distortion of the intestine. He describes four cases of stercoral ulceration due to impaction of masses of meconium in the bowel. He suggests that the obstructing ano-rectal plug is related to immaturity of the colonic rectal expulsive mechanism, and that intestinal meconium plugs and stercoral ulceration of the large bowel appear to be related to localized abnormalities in the water balance in the affected segment of the bowel.

R. B. ZACHARY (*ibidem*) describes 10 cases in which obstruction of the large bowel in the first few weeks of life was due to impaction of the bowel contents. Four of these were examples of meconium plugs. Relief was given by rectal examination and rectal wash-out. In the other six cases there was incomplete obstruction of the colon by hard faecal matter between the ages of four days and five weeks, and perforation of the bowel occurred in all cases.

P. P. RICKHAM (*ibidem*) describes a single case in which large numbers of small calcified masses occurred in inspissated meconium in a new-born infant. Intestinal obstruction resulted, and despite operative relief of the obstruction with cleansing of the bowel, the infant died. The detailed care of the infant during the three weeks in which it was kept alive is described.

Oesophageal Reconstruction.

C. D. SHERMAN AND D. WATERSTON (*Arch. Dis. Childhood*, February, 1957) describe their experiences in the reconstruction of the oesophagus in children by means of transplantation of portion of the colon into the thorax. The chief indications have been atresia of the oesophagus with or without tracheo-oesophageal fistula, and strictures of the oesophagus resulting from the swallowing of corrosives or from acid reflux into the oesophagus through an incompetent cardia. Although they have had no cases in which the lower part of the oesophagus has been resected, for example

because of bleeding varicosities, they point out that there is a growing opinion among surgeons that the oesophagus should almost never be anastomosed to the stomach. If this resection is carried out, the interposition of a loop of colon between the lower part of the oesophagus and the stomach is probably wise. The two methods of reconstruction have been used. The first involves the transplantation of the transverse colon iso-peristaltically into the left posterior quadrant of the chest on a pedicle containing the left colic artery. The operative details are described. The method has the advantage of preserving intact the all-important cardiac sphincter mechanism between the oesophagus and the stomach. The second method of reconstruction uses the ascending colon transplanted iso-peristaltically into the retrosternal position. The advantage of the first method is that, because the normal oesophago-gastric junction is preserved, there is no danger of later peptic digestion of the colonic transplant with perforation. The main disadvantage to the first procedure is the fact that the blood supply must be brought through the diaphragm, and considerable care must be taken to make sure that the opening in the diaphragm is neither so large as to result in a hernia nor so small that it causes vascular obstruction. The advantage of the second method lies in the fact that the anterior mediastinal route seems a very satisfactory one, because it is the shortest distance to the neck, and because it does not interfere with the blood supply to the transplant. The authors state that reconstruction of the oesophagus by colon transplant is still in the developmental stage, but sufficient experience has been accumulated to suggest that further trial and evaluation are worth while. They admit that several years of further observation of the present series of patients will be necessary before they can with certainty justify their initial enthusiasm about this procedure.

Gastro-enteritis in Infancy.

R. J. DERHAM AND M. M. ROGERSON (*Am. J. Dis. Child.*, February, 1957) review the occurrence of gastro-enteritis in infancy in Liverpool, England, over the ten years from 1945 to 1955. They find that over this period there has been a steadily decreasing mortality rate, which they attribute to a number of causes. The first is improved understanding and management of blood electrolyte disturbances. The second is the great advance that has been made in the bacteriological studies of pathological strains of *Escherichia coli* and their relationship to gastro-enteritis. The third is the use of antibiotics. The fourth is the overall improvement in housing and hygiene, the greater number of medical workers available, and the greater awareness of the important aspects of the problem among the public and among doctors. The report emphasizes the part played by *E. coli* in causing gastro-enteritis in infants. The authors survey in detail the bacteriological studies of infants admitted to hospital in Liverpool during the period from March to August, 1955, suffering from this disease. Of

466 cases, pathogenic strains of *E. coli* were isolated and were regarded as the cause of the disease in 170, while *Shigella sonnei* was found in 97, *Salmonella typhimurium* in 30, and mixed infection with these three groups of organisms in 23. In the remainder no pathogenic organisms were isolated. This prominence of *E. coli* in patients under the age of two years is very plain. The authors point out that this organism is not blamed in all centres in England and not commonly blamed in America, and they suspect that the failure to recognize it may be due to a lack of awareness of its importance or to pre-occupation with the search for other organisms. *E. coli* is of the greatest importance in relation to younger infants, and their susceptibility is so high that cross-infection easily occurs. Of the pathological strains of *E. coli*, 0.111 was found most commonly. Next came 0.119 and less often 0.115, 0.26, 0.128 and 0.86. The strain 0.111 was found to be the most dangerous, and 0.119 seemed to be the strain that was most often the cause of cross infection, and sometimes it proved very difficult to clear from the infants' stools.

Infantile Optic Atrophy.

J. H. DOGGART (*Trans. Ophth. Soc. U. Kingdom*, 1955) discusses the diagnosis of optic atrophy in infancy. Pallor of the disk, the cardinal sign of optic atrophy, is common to all types. However, disk pallor alone does not necessarily mean optic atrophy. The average infant's disks are paler than those of children and adults. Also, the possibility of delayed myelination must be borne in mind. A few infants who are supposed to show partial atrophy are examples of imperfect development, i.e., hypoplasia, congenital holes, crescents and unilateral myopia can modify the colour of the disks. Obscuration of the physiological cup may not necessarily mean secondary optic atrophy; the presence of vestigial veils of connective tissue may confuse the issue. Blurred margins without corroborative signs of disease should not be viewed with alarm. Developmental excavation of the disk has been confused with glaucoma; other features will make differentiation possible. Once a diagnosis of atrophy has been made, the prognosis must be guarded—some babies with apparent atrophy will subsequently be found to have normal vision. In others, mental development may help overcome the visual disability.

The Treatment of Intussusception in Infants and Children.

G. PACKARD AND R. ALLEN (*Surgery*, April, 1957) have treated 100 patients with intussusception in the Denver Children's Hospital during the ten years ending January 1, 1956, with only one death. Twenty-six patients were treated successfully with a barium enema alone, 29 were treated with a preliminary barium enema followed by surgery, and 43 were treated by surgery alone. The authors consider that appendicectomy apparently carries no added risk. Recurrence was rare, but slightly more common after closed reduction, while late adhesive obstruction was seen in two cases after operation. They state that treatment

with a barium enema has resulted in sufficient successes during this period, so that its use as a primary method of diagnosis and treatment has been on the increase. Its value is greatest in the early cases, and surgery must follow if there is any doubt at all of its results. In late cases surgery is the logical resort.

Hypothermia in General Pædiatric Surgery.

D. BRAYTON and G. LEWIS (*Ann. Surg.*, March, 1957) state that mild hypothermia is a safe and useful adjunct to prepare acutely febrile children for emergency operations, or normothermic children for elective operations of long duration. They present a preliminary study of a small series of cases intended to demonstrate that this is so.

Effect on the Rabbit Fœtus of Administration of Propylthiouracil to the Mother.

E. KREMENTZ, R. HOOPER and R. KEMPSON (*Surgery*, April, 1957) have studied the effects of propylthiouracil and propylthiouracil and thyroxin on the rabbit fœtus when administered to the pregnant adult, determining the average weight of the fœtuses and the relative weight of the fetal thyroids as compared to normal controls. They have found that there was a significant relative weight gain in the fetal thyroids of both groups, the addition of thyroxin to propylthiouracil causing no significant change in the relative fetal thyroid weights as compared with those resulting from the administration of propylthiouracil alone. A definite decrease in the average total fetal weight was noted when the adult received either propylthiouracil or propylthiouracil and thyroxin, although the decrease in weight was less when thyroxin was added. From clinical experiences reported from the experimental findings, the authors consider that pregnant women with thyrotoxicosis should be controlled whenever possible by surgery after the euthyroid state has been attained with the minimal amount of antithyroid drug administered in conjunction with thyroxin.

ORTHOPÆDIC SURGERY.

Perilunar Dislocations.

C. J. WAGNER (*J. Bone & Joint Surg.*, December, 1956) has studied 78 carpal injuries each with some degree of loss of the normal relationship between the lunate and capitate bones. These are classified generally as "perilunar dislocations". They are correlated from the clinical viewpoint of treatment and prognosis. Functionally the lunate, triquetrum and pisiform act as a proximal unit. The trapezoid, capitate and hamate form the distal unit. The navicular forms a connecting rod between the two. The trapezium functions as a modified metacarpal. The author discusses the mechanics of the wrist joints and movements in detail, and then uses his conclusions in explaining the mechanism of wrist fractures. Thus forceful dorsiflexion of the wrist can easily fracture the navicular. The lunate is buttressed by the dorsal lip of the radius; if this dorsal buttress gives way, or there is no volar

inclination of the distal surface of the radius, or the force is applied perpendicularly to the hand, the entire carpus is dislocated, the relationship between the triquetrum and lunate on one side and the lunate and navicular on the other being disturbed. This type of displacement is rare. If the navicular is fractured, usually the carpal bones maintain their normal relationship; but if the force acting continues, then the fragments of the navicular separate, the proximal pole remaining in normal relationship with the lunate and the end of the radius. The distal pole remains with the rest of the carpus, which is dislocated dorsally. If the navicular is not fractured, then it may either be dislocated with the carpus or remain with the lunate. Usually it is dislocated with the carpal bones, the lunate alone being left in relationship to the end of the radius. A forward dislocation of the lunate may follow the dorsal carpal dislocation. The carpus assumes a normal position at the end of the radius. This may be distinguished from a simple volar dislocation of the lunate by the mobility of the carpus during reduction. The last type is dislocation of the carpus with a fractured lunate, a fragment remaining attached to the navicular. Finally, the author draws attention to the perilunate trans-navicular type with reduction of the dislocation, the navicular fragments being left incompletely reduced. This fracture does badly when treated by all the usual methods, and he advises early arthrodesis. Unreduced perilunate dislocations without fracture of the navicular can be treated by open reduction with satisfactory results. When the lunate is dislocated, it should be reduced by closed manipulation if possible. Excision of the lunate gives a weak wrist. Primary arthrodesis is advocated when the lunate is fractured, as aseptic necrosis of this bone is common.

Partial Synovectomy and Curettage in Tuberculosis of the Hip.

M. C. WILKINSON (*J. Bone & Joint Surg.*, February, 1957) presents a supplementary report of the treatment of 39 patients with tuberculosis of the hip. None had destruction of the joint space. They were treated with general measures, the administration of antibiotics and intra-articular operation. This varied between simple arthrotomy and partial synovectomy with curettage of bone foci. Operative treatment was carried out earlier in the later cases of this series, as the author became more confident in the response to antibiotics. Some mobility was achieved and the disease was controlled in 31 out of the 39 cases. Operation to stabilize the hip was necessary in eight. Three of these failures were in adults and one was in an adolescent. Also, it was noted that five failures occurred before 1951. Hence the earlier and more radical excisions were better. The disease progressed despite this regimen in only one case, with final complete destruction of the hip joint; this patient was a child. The two patients who relapsed were both children operated on in 1950. An acetabular cavity was not curetted. Early progress was good, but the cavitation increased in size. In one case the hip was fused, in the other the cavity was curetted out. The hip finally func-

tioned well. The author discusses the pathological findings at operation and the prognosis. He states that radiographic changes usually indicate severe involvement and poor prognosis, but that an absence of radiographic changes may not mean a good outlook, as extensive synovial disease may be present. This type of case usually presents with gross edema and spasm of the hip joint. General and local improvement follows operation. Associated lesions in the lungs or elsewhere decrease, and the erythrocyte sedimentation rate falls. Late changes occur in the joints, which recover. The femoral head becomes enlarged, and lengthening also occurs. The head may not completely fit in the acetabulum.

Persistent Fœtal Alignment of the Hip.

E. W. SOMERVILLE (*J. Bone & Joint Surg.*, February, 1957) draws attention to rotation deformity of the lower limb. He states that fetal alignment is an anteversion of some 60°. During growth this is usually moulded away. If the anteversion is retained or increased, subluxation and dislocation of the hip often follow. This is because internal rotation of the hip occurs with extension, owing to the pull of the hip joint ligament. This tension through the ligament usually leads to the corrective moulding. Should this fail and the capsule does not stretch, then the child's legs will not externally rotate when they are extended. Compensatory external rotation of the lower part of the leg occurs, accompanied by eversion of the feet with flattening of the longitudinal arch. This condition is noted more often in girls, who have congenital dislocation of the hip more frequently than boys. It is also commonly found in the good leg when the other hip is dislocated. In the X-ray film, an obtuse angle seen between the neck and the shaft is due to the anteversion. Treatment is by rotation osteotomy in the subtrochanteric region, with plating.

Slipped Femoral Epiphysis with Severe Displacement.

C. H. HEYMANN *et alii* (*J. Bone & Joint Surg.*, April, 1957) describe a conservative operation for slipped epiphysis. They recommend the careful piecemeal removal of the buttress of the antero-superior part of the femoral neck, through a Smith-Petersen incision. This allows great improvement in the range of movement of the hip joint. The present management of severe deformity following the slipping of a femoral epiphysis is usually by subtrochanteric osteotomy. External rotation is corrected. The other two methods—namely, open reduction through the epiphyseal plate and wedge osteotomy of the femoral neck—are commonly followed by avascular necrosis of the head. The authors discuss the outcome of conservative management, including the realignment that occurs with growth. They selected for operation patients who presented with severe displacement of the epiphysis and a disability that demanded assistance. This conservative operation was described in 1909 by Whitman and later in 1919 by Stöfel, but the authors performed it without prior knowledge of these papers.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on May 21, 1957, PROFESSOR W. K. INGLIS in the chair. The principal speaker was DR. H. M. WHYTE, Director of the Clinical Research Department.

Clinical History.

The following clinical history was presented.

A woman, aged 58 years, of "Tahitian descent", was a patient in another hospital, where she died. Because of a feeling of racial isolation the history was difficult to elicit. Nothing was known of her childhood. At the age of 18 years she was said to have been admitted to hospital two weeks before the birth of her first child. No details of this admission could be traced, but the pregnancy was normal. Six years later she had another normal pregnancy at the same hospital. Examination of her urine at that time showed it to have a specific gravity of 1.020; albumin was not detected. No other details were recorded. When she was aged 47 years, two grandchildren with whom she was closely associated developed positive Mantoux reactions. One of these children had an opaque area in a lung. About one year before her death the patient came under the care of her attending physician, complaining of weakness of the legs and breathlessness on exertion. The blood pressure was 160/120 millimetres of mercury. She had auricular fibrillation and oedema of the ankles. She responded well to treatment with mersalyl, with diuresis and loss of weight. Four months later she developed fairly sudden right-sided pulmonary consolidation with fever. She was treated with a tetracycline and responded well, with resolution of chest signs and fever.

After this illness swelling of the legs reappeared and extended as far as the hips. She was now treated with twice-weekly injections of mersalyl, and the oedema disappeared. She was weak and breathless for several months. She complained of leg pains and parasthesia at night. She now commenced to suffer from diarrhoea. She had had severe hemorrhoids, and with the onset of diarrhoea became at times incontinent of faeces. Her children thought her complexion was becoming darker. She had been in the habit of drinking alcohol equivalent to two schooners of beer daily, and she drank a lot of water. There was no frequency of micturition and no difficulty in passing urine. She was admitted to hospital under the care of a consultant, complaining of weakness, lethargy, diarrhoea and soiling of the bed. She was dark-skinned with grey hair. There was considerable pigmentation of the palms and of the oral mucosa. The nail beds and conjunctivae were pale. She had lost weight, as evidenced by folds of redundant abdominal skin. The pulse was small and regular (rate 75 per minute), and the central nervous system was normal, as was the respiratory system except for some evidence of bronchitis. Rectal examination showed external hemorrhoids, a lax anal ring, and an ill-defined boggy mass or thickening thought to be in the wall of the rectum or in the tissues around it. The urine had a specific gravity of 1.008 and was acid and clear, containing no albumin. The haemoglobin value was 8.5 grammes per centum. The white cells numbered 7200 per cubic millimetre (the differential count being normal). Examination of the red cells revealed a few microcytes and macrocytes. The serum electrolyte values (in milliequivalents per litre) were as follows: sodium, 130; potassium, 4.7; chloride, 105; carbon dioxide combining power, 19. The interpretation was hyponatraemic acidosis.

Treatment with "Probanthine" and *Mistura Catechu cum Opio* was commenced and a blood transfusion was given. After several false starts a Kepler test was performed with care. The results were as follows: overnight urine volume (10.30 p.m. to 7.30 a.m.), 24 ounces; 40 ounces of water drunk at 7.30 a.m.; volume of urine at 8.30 a.m., four ounces; at 11.30 a.m., five ounces; at 9.30 a.m., two ounces; at 12.30 p.m., four ounces. These results were regarded as abnormal. An X-ray examination of the chest showed opacities at the right mid-zone. The diarrhoea was partly relieved by treatment. Cortisone (25 milligrammes three times a day) was given in the nature of a diagnostic test, and after nine days the consultant remarked that no outstanding amelioration of the condition had occurred. The diarrhoea became worse, and the patient was often incontinent of faeces. After three weeks the patient's condition was reassessed. A blood count now showed that the red

blood cells numbered 3,520,000 and the white blood cells 5900 per cubic millimetre (neutrophils 65%, eosinophils 6%, basophils 1%, lymphocytes 27%, monocytes 1%). The packed cell volume was 35%, the mean corpuscular volume was 90, the mean corpuscular haemoglobin was 34%, and reticulocytes numbered 1%. The red cells were variable, and many macrocytes and microcytes were present. The leucocytes were mature, and hypersegmentation was a feature of some of the neutrophils. Platelets were normal. The Mantoux test gave a negative result. The zinc sulphate turbidity was one unit; the serum bilirubin content was 0.25 milligramme per 100 millilitres; the serum alkaline phosphatase was seven units; the serum protein content was 4.1 grammes per centum (albumin 1.8 grammes, globulin 2.3 grammes). Urine examination now showed the specific gravity to be 1.010; the urine was acid and contained one-third albumin. Vitamin B₁₂ therapy was commenced. After five, seven, eight and eleven days the reticulocytes numbered 0.4%, 1.5%, 0.9% and 1.8% respectively. Diarrhoea and faecal incontinence continued. A barium meal passed to the colon in 24 hours, and X-ray examination showed no abnormality in the mucosal pattern of the small intestine. Numerous urine examinations showed average figures of specific gravity 1.010, albumin one-third; the urine was acid.

Oedema of the feet now reappeared. There was no oedema of the sacrum and no evidence of venous thrombosis. Numerous pus cells and non-haemolytic streptococci were found in the urine. Several notes of poor urinary output were made; but the average intake of fluid was 40 ounces and the output 30 ounces. The faeces were fluid or semi-formed and foul-smelling.

Ten weeks after her admission to hospital the patient had a pain in the chest, thought to be a coronary occlusion or pulmonary embolus. It passed off, but recurred daily for a week. During the last attack the blood pressure fell suddenly to 55/30 millimetres of mercury, and the pulse was imperceptible. She died next day. The erythrocyte sedimentation rate a few days before her death was 56 millimetres in one hour (Wintrobe).

Clinical Discussion.

PROFESSOR W. K. INGLIS: I will ask Dr. Malcolm Whyte to discuss the case.

DR. H. M. WHYTE: For various reasons my preparation of this discussion has been disturbed, and up to a day or two ago I was very undecided as to how to present it to you. That is largely, I am afraid, because of ignorance of the diagnosis. I have ended up just letting my thoughts flow, and here they are. But first I must say that the protocol tells a long and complicated story. It is confusing, and some of it is probably quite misleading. So let us put it aside and start afresh, ridding our minds of all the thoughts, suggestions and possible diagnoses which the doctors in charge of this patient have written in between the lines of the medical record. Let us sit by her bed, as it were, towards the end of her illness and review the position. And what we do not know, we shall make up, in accordance with the licence accorded to poets, Press reporters and members of our august profession.

So here she is, a grandmother, 58 years of age, with grey hair, a crooked nose and brownish skin, as we can see by the photograph. She complains mainly of weakness and lethargy and diarrhoea. The diarrhoea is most annoying and distressing. It has been troubling her for at least a couple of months now, and has made her piles worse, and at times she is unable to control things, so that she soils her bed. The motions are fluid or semi-formed, foul-smelling and brown, and show no evidence of abnormal matter. I think we should look at more of her stools for signs of blood and mucus. She does not admit to any attacks of dysentery during her earlier days, nor can we get her to give us a clear account of her bowel's behaviour before this present illness. However, it is quite clear that she was weak and breathless for some months before the diarrhoea started, so we cannot attribute those symptoms to exhaustion or to potassium loss due to diarrhoea. I wonder why she has this looseness of bowel action. We should repeat the digital examination of the rectum, and use a proctoscope, as well as looking closely at the faeces, before searching too far in the direction of the dysenteries, ulcerative colitis, malabsorption states and so on. The resident medical officer thought that the anal ring was lax, and that there was a peculiar thickening and boggy mass about the rectal wall.

Where are we? Diarrhoea for two or three months, weakness, lethargy and breathlessness for longer; oedema, too. In fact, she has not been well for more than a year. In the beginning it was weakness of the legs, increasing breath-

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lessness when she exerted herself and swelling of her ankles. A letter from the doctor she consulted states that she had hypertension and atrial fibrillation at that time, and that she improved when treated with a mercurial diuretic. This sounds like cardiac failure—hypertensive failure. Why was she hypertensive? Maybe it was ordinary essential hypertension; she knows nothing of her family history to help us on this score. Her two pregnancies seem to have been uneventful. We can get nothing from questioning her to suggest acute nephritis at any stage, or pyelonephritis or any other renal condition. Nor is there anything to indicate the possibility of pheochromocytoma, or *polyarteritis nodosa*, or chromaffinoma, or Cushing's disease. Could this temporary episode have been thyrotoxicosis? We had better check her thyroid and, to be on the safe side, her femoral pulses as well. I wonder, too, what was found in her urine at that time, one year ago.

Anyway, she remained fairly well for four months, at least not oedematous, presumably having regular injections of mersalyl. She then suddenly developed a cough and fever with signs in the right side of her chest which quickly resolved after treatment with an antibiotic. But oedema recurred, worse than before, right up her thighs, and the weakness was worrying, the breathlessness distressing, and there were pains and pins and needles in her legs at night; that is when the diarrhoea started.

Now the daughter has told us that her mother's skin has been getting darker. Admittedly she has a touch of Tahiti in her blood; but it is especially interesting because she has these dark pigmented areas in the mouth and her palms are pigmented, she has lost weight and her blood pressure is now lower than normal. What does that suggest? Addison's disease. That could account, too, for the weakness, lethargy, apparent anemia, diarrhoea and, through increased liability to infection, the pulmonary episode of a few months ago. How else could we account for these features? *Anorexia nervosa* is not in the picture; she is not extremely wasted or cachectic; there is no fever to suggest a grumbling infection, like tuberculosis; and there are no enlarged glands or abdominal organs or anything else to suggest neoplasia, Hodgkin's disease or the like. Salt-losing nephritis can mimic Addison's disease. Usually it is chronic pyelonephritis, and I notice that she has heavy albuminuria, that the specific gravity has always been about 1.010 and that numerous pus cells are to be seen in the centrifuged deposit. So she has a renal lesion. Addison's disease cannot cause that.

Perhaps both organs, kidneys and adrenals, are diseased. It makes it difficult to decide. For example, we can give a water-load, knowing that patients with Addison's disease will absorb it perfectly well, but are usually unable to excrete it at all rapidly. But, unfortunately, exactly the same result may be obtained in renal disease as in dehydration, cachectic states, thyroid disorders, steatorrhoea etc. This simple test was used with this patient, the first part of a Kepler test, and although the result was positive—that is, the volume of night urine, 24 ounces, was greater than any of the hourly volumes excreted after a large water-load—it is not helpful in deciding about Addison's disease because of the chronic nephritis. Part two of Kepler's test required the services of a laboratory; but even then a positive result might be expected with either Addison's disease or renal disease. How else can we decide? If there was no renal trouble, then we could treat the patient with cortisone and repeat the water test. In pure Addison's disease the ability to handle water is restored by treatment with cortisone (though not by desoxycorticosterone acetate) and, of course, other symptoms and signs might slowly diminish. If we had the salt in urine measured, then treatment with DOCA would cause it to diminish in Addison's disease, but not in salt-losing nephritis. For the best test you would have to come to Sydney Hospital to someone like Dr. Stacy, who would measure the amount of adrenal cortical hormones excreted in the urine before and after treatment with an intravenous drip of ACTH to find out whether there is any adrenal tissue present capable of being stimulated. This would be the best way of settling the question of Addison's disease.

So we do not know about Addison's disease, but it is a distinct possibility. About half the cases are due to tuberculosis, most of the others to atrophy for no apparent reason, a few to amyloidosis, neoplastic infiltration, syphilis, and arterial and venous blockage. Is there any evidence of tuberculosis? Yes, the patient was closely associated with her two grandchildren when they were infected—perhaps she infected them—eleven years ago. Perhaps her lung condition, eight months ago, was tuberculous; and she still has some noises in her chest. We should have her chest

X-rayed, her sputum examined and a Mantoux tuberculin test done. A radiograph should be taken of the adrenal areas, in a search for calcification.

And what of the renal lesion? Heavy albuminuria is the outstanding feature, with kidneys so badly damaged that they apparently have very little control over specific gravity. Such a loss of albumin could lead to hypoproteinaemia and the nephrotic syndrome, and this could have played a major part in the oedema she has had on and off. In fact, this is a more likely cause of oedema than cardiac failure in her last months, since she had no evidence of pulmonary congestion, and no one has found systemic venous engorgement or an enlarged liver. She has nephrosis. Nephrosis can come out of the blue as type II nephritis, or it can follow glomerulonephritis, or arise in diabetes or syphilis or be due to drugs like gold, or to *lupus erythematosus*, amyloidosis, multiple myeloma, pyelonephritis, or thrombosis of the renal vein, or other causes of renal venous congestion such as cardiac failure. In the present case we have no evidence for most of these. Lupus would help us to explain the multi-system affections, the temporary cardiac condition of a year ago, the more recent pulmonary episode, and the renal trouble and the diarrhoea; but there has been no rash, no joint involvement and no fever of note. Lupus does not seem to attack the adrenals, but it has been known to lead on to amyloidosis, which in turn could do so. Venous thrombosis is another possibility which could occur spontaneously, or in conjunction with any other disease, and give a fluctuating picture such as we have here. Spread of the thrombus to involve the inferior vena cava could have caused the recent extension of oedema. The absence of dilated by-pass veins over the abdomen does not rule out this condition. Nor is there any real evidence of constrictive pericarditis or anything else causing venous congestion.

Pyelonephritis is, of course, a common disease, but it does not often cause nephrosis. This woman has pyuria; but this in itself tells us very little. I am presuming that a clean catheter specimen was taken; at any rate, the procedure should be repeated and an attempt made to grow organisms. Secondary infection is common in chronic renal disease of any cause.

However, she could have had progressive renal failure from pyelonephritis for some years—ordinary pyelonephritis, or perhaps tuberculous renal disease. If it is tuberculous, there should be some primary lesion elsewhere, perhaps in the lung, and it is surprising that there is no haematuria, no pain and no suggestion of bladder involvement. Previous hypertension could have been renal in origin, and present hypotension (if not due to extra-renal disease) could have been caused by loss of salt, perhaps accelerated by mersalyl. Renal disease could also be responsible for her weakness, lethargy, breathlessness, oedema, anemia and perhaps diarrhoea. But if tuberculosis is to be considered, then we must also think of amyloid degeneration. Amyloidosis could also complicate syphilis, lung abscess or other chronic suppuration or multiple myeloma, but there is no evidence of these. Of course, amyloidosis can also be primary, and as such is known to cause weakness, lethargy, loss of weight, pulmonary symptoms, oedema, paraesthesiae, diarrhoea, and even laxness of the anal sphincter. We could imagine involvement of the kidneys, the adrenals, the gut and even, perhaps, the heart. So amyloidosis is quite a possibility.

I have not thought of anything else important that I should be discussing, and if the pathologists care to put up anything else, I suggest they have the cases confused. In summary, I believe there is renal disease and perhaps Addison's disease. The underlying cause may be tuberculosis, perhaps with amyloidosis. Or these organs may be affected by amyloidosis alone, which may be primary or secondary to disease elsewhere—tuberculosis, syphilis, myelomatosis. There is the vague possibility of disseminated *lupus erythematosus*, again perhaps with amyloidosis. Thrombosis of the renal veins may be accounting for some of the story, and we can never be too certain of omitting neoplasm, perhaps of the bowel, with secondary deposits elsewhere.

So far, you may notice, I have confined my discussion to facts which can be elicited from the history, examination and simple tests in the ward or home, without recourse to laboratory aid. Perhaps, out of courtesy, we should briefly thumb through the results of investigations. So far as Addison's disease is concerned there is nothing to help us—the serum sodium content is low and the potassium content normal, and the radiographs do not show any calcification in the adrenal areas. In relation to renal disease, the serum sodium content is probably depleted, there is not much potassium retention (perhaps thanks to diarrhoea), but the

acidosis is significant, and the blood urea level, had it been estimated, would probably have been high. Hypoproteinaemia is confirmed, and the absence of any clinical evidence of an hepatic cause for this is strengthened by the normal serum bilirubin and alkaline phosphatase contents and the normal results to flocculation tests. Culture of the urine was not helpful—at least no tubercle bacilli were found; the organism grown could have indicated renal infection, old or new, or it could have been a contaminant.

And what pointers have we to tuberculosis? The X-ray film shows that she has been infected, for there is an unmistakable Ghon's lesion, so the negative response to the Mantoux test can be disregarded; perhaps the cortisone had made it negative temporarily. As for the lesion in the mid-zone of the right lung, I think I shall leave that for you to discuss; but there is calcification at its root, which must be very suggestive of tuberculosis at some stage. There is no calcification in the region of the adrenals, or in the kidneys. What else is there? The blood count is not really helpful. The erythrocyte sedimentation rate is raised; but this could have been due to terminal myocardial infarction or to the nephrosis itself. An electrocardiogram taken after the chest pain incident could conceivably indicate a posterior infarction, I am told; but extremely low voltage is the main feature, and would be compatible with any general myocardial weakness as in Addison's disease or amyloidosis. Finally, there are barium studies of the bowel, which seem to prove very little except that this is a very sophisticated method of discovering impacted faeces in the rectum. This apparently faecal mass was present in December, and it was still present in January—one month later. It remained even when the barium drained out around it. Perhaps it is a neoplasm or a mass of some other sort; but I like to think it is faeces—the "ill-defined boggy" felt by the examiner's finger.

So our so-called special investigations have added nothing to our previous list of possible diagnoses, except impacted faeces. Finally, it is possible, of course, that the patient had a myocardial infarction which terminated her life.

What would I do? I have mentioned during my discussion what I would do by way of extra interrogation, physical examination and special tests to get further clues about Addison's disease, tuberculosis and the rectal contents. We could think about other tests, if necessary, after these had been done. The diagnoses most likely to fit this story to my mind are: chronic renal disease and Addison's disease due to tuberculosis, perhaps with amyloid degeneration, and impacted faeces and terminal myocardial infarction. At any rate, there is plenty for you to discuss.

PROFESSOR INGLIS: Have we a radiologist here? Because I think that before we throw the subject open for general discussion, it might be desirable—not that I wish to cast doubt on the radiological interpretations of Dr. Whyte—to have a radiologist's opinion on these films.

DR. J. LYALL: The chest X-ray film shows a primary focus as Dr. Whyte mentioned, and an area of calcification at the right hilum. The lateral view shows that the area of calcification is indeed at the hilum, and it is very likely calcification in lymph glands. The lesion lateral to the right hilum is smooth in outline and lies above the horizontal fissure. It is probably attached to the horizontal fissure, as the fissure is tented towards its medial end. The antero-posterior film taken approximately six weeks later still shows this lesion with a smooth outline, and it has the appearance of a soft-tissue mass. It is almost a true lateral, so that the lesion appears to be extrapulmonary. It is most probably a thickening of the pleura in this area.

The cardiac outline is not remarkable. There is some prominence of the aortic knob and some dynamic unwinding of the arch of the aorta. There is no evidence of calcification in the heart shadow.

Commenting on the barium studies: no abnormality is seen in the stomach; four hours after the meal there is some residue in the stomach. The barium enema examination was made approximately a month earlier. In both enema films there is a mass in the rectum with a smooth outline and with flecks of barium through it. It has the appearance of impacted faeces with the enema flowing round it. Barium in the rectum in the film of the barium meal at four hours is probably barium remaining from the enema of a month before, unless another enema had been attempted in the interval. The series of films up to seven days after the meal shows an unusually large amount of barium remaining in the bowel. A possibility to account for this would be a fistula between small and large bowel to allow a recirculation of the barium. However, seeing that it has

taken so long for the rectum to be cleared of barium after the enema, it seems reasonable to say that the barium in the bowel here is due to stasis.

DR. W. L. CALOV: I do not think that I place great reliance on the buccal pigmentation as an indication of the presence of Addison's disease in this case. I do not know a great deal about Tahitians, but I have seen great numbers of Melanesians, and if they have a lesion of the mouth like an ulcer, or pyorrhoea or a wound of the mouth, it is likely to become pigmented. So I think that the pigmentation could be entirely discounted. You sometimes see a Melanesian who has had pyorrhoea, and he has a classical lead line on the gums—a blue line. So every time you see a blue line, it is not necessarily due to exposure to lead.

I am interested in the X-ray films of the thorax. The second one shows the cardiac outline a bit widened. This might be due to the position of the diaphragm to some extent; but I rather take it that it is due to some degree of cardiac dilatation, from cardiac failure. The electrocardiogram I do not think shows anything specific. It just really shows that she has a "crook heart".

The lesion in the right mid-zone looks as though it could be a segmental collapse. That could be a possible source of her trouble. There could be a little bronchogenic carcinoma there, slowly growing, and she could have secondary deposits elsewhere; unfortunately not revealed by any of these examinations. I am at a complete loss in regard to the diagnosis, unless it is something perfectly simple like chronic nephritis and congestive cardiac failure. I think it might be boiled down to that in the long run. Of course, we know she had constipation, but I do not think constipation caused her death.

DR. W. EVANS: I, like Dr. Calov, find considerable difficulty in elucidating a diagnosis. Dr. Malcolm Whyte has given us an extraordinarily clear exposition of the whole case, and it seems to me that the three major probabilities are as he has put them—Addison's disease, renal disease and possibly cardiac failure. Addison's disease is difficult to exclude completely, because of the history, the previous existence of pulmonary tuberculosis and the hypotension, although there is no definite proof. So although tuberculosis is a probability, I do not think it can be accepted as proven.

The possibility of renal disease is definitely present in view of the very marked albuminuria, and I noted that, in the early stage at least, some of her symptoms were due to cardiac failure. Also her final end suggested that she had a myocardial infarction. However, in her later history she developed very heavy albuminuria with pyelitis, and pus cells in the urine, so that at that stage at least she must have had some nephritis.

The possibility of an alcoholic cause presents itself to my mind. It has not been mentioned up to date. She had mentioned that she drank two schooners of beer a day. Well, a person who admits to two schooners quite often exceeds that amount. So that the possibility presented itself to me as the cause of her cardiac failure and her hypoproteinaemia. Against it, of course, is the fact that the heart is not very large, but still it can be accepted as a possibility. She did have cardiac failure, and alcohol cannot be excluded as the cause.

To summarize my point of view, I think that the possibility of Addison's disease exists. I think she had cardiac failure and the possibility of a nephritic element as well.

DR. G. E. BAUER: There are several things that I should like to mention about the electrocardiogram, which I saw very briefly. I think it does show a generalized myocardial disease. I am not quite sure of the date of this electrocardiogram. Was it taken before this final episode? No. It was after the chest pain. The main thing to be seen in this electrocardiogram is the fact that the heart was grossly clockwise-rotated. The right ventricle comes round towards the front, which is, of course, the sort of thing that you see in a right-sided enlargement. With one little further excuse of not having seen the protocol before and of having arrived late, I must gracefully retire.

DR. P. F. HALL: As the protocol suggests, this case raises certain questions of interest related to tests of adrenocortical function. It is, I think, important to bear in mind that the adrenal cortex secretes hormones of three types—glucocorticoids, mineralocorticoids and androgens. At all events, these are the only cortical hormones the secretion of which can be subjected to direct or indirect measurement.

Glucocorticoids (principally hydrocortisone) have the property of assisting the body to excrete a water-load. The

mechanism of this property is uncertain. Some workers believe that glucocorticoids affect the rate of secretion of antidiuretic hormone; others hold that these hormones affect renal blood flow. In either case, the Kepler test (or one of its several modifications) attempts to measure this property of glucocorticoids. Such measurements are indirect and at best crude. The test is one of potential secretion, in contrast to tests which measure the basal level of cortical activity.

In this particular patient the test proved of no value, for the reason that patients with oedema or those who have recently exhibited oedema are quite unsuitable for water-load tests. Renal disease and cardiac failure are among those conditions which interfere with such investigations. A more satisfactory test is that which Dr. Whyte mentioned—namely, comparison of the response to a water-load before and during cortisone therapy. This is hardly a test of adrenocortical function as such, but is a valuable aid to the diagnosis of Addison's disease. Such a procedure is much more valuable than the subjective assessment of symptoms before and during the administration of cortisone.

Pigmentation of the oral mucosa and palms of the hands is normal among the dark races, especially Negroes. Unless it can be shown that the pigmentation appeared during the present illness, this sign cannot be seriously considered as suggestive of Addison's disease. In short, the evidence as presented does not suggest Addison's disease, although this condition cannot be entirely ruled out.

DR. B. P. BILLINGTON: I can throw no light on this case at all. The patient had apparently a colonic obstruction with diarrhoea. The diarrhoea was of a foul-smelling nature. Apart from faecal impaction, which undoubtedly was present, one other possibility that might cause this continuous diarrhoea is a pelvic abscess. She did not have leucocytosis. It certainly could account for the boggy which was felt *per rectum*. The other possibility is that a foul rectal discharge may occur with a chronic enteritis, sometimes particularly in the terminal stages of tuberculous enteritis.

The question I should like to ask does not concern the alimentary state, but the renal situation. Early in the picture, she had oedema, but no albuminuria, and a haemoglobin value of 8.5 grammes *per centum*, which to my way of thinking is a severe anaemia. It could be on the basis of chronic nephritis; but I just wonder about that in the absence of oedema, and a clear urine as far as albumin was concerned. I cannot understand what was going on at that stage, and I do not understand what was the matter with her at all.

DR. B. D. STACY: I do not think there are very many comments that I can make. Dr. Whyte and Dr. Hall have very ably pointed out the pitfalls that may occur in the interpretation of the Kepler test. Since the second part of the test was not carried out, comment must be limited. The Kepler test may give false-negative results in cases in which adrenal insufficiency is of recent origin, or in which there is a bare minimum of adrenocortical tissue still functioning. Much more common are the cases in which the test falsely indicates the presence of Addison's disease. Dr. Whyte and Dr. Hall have dealt with these cases. In view of the number of falsely positive results that may be obtained with the Kepler test, the tendency nowadays is to place more reliance on other tests of adreno-cortical function, and I thank Dr. Whyte for his kind reference to some work that we have been doing in this direction.

We keep in mind the various aspects of the adrenal cortex as outlined by Dr. Hall. By measuring the urinary excretion of 17-ketosteroids, 17-hydroxycorticoids and electrolytes before and after ACTH administration, we are able to obtain direct evidence on the functional status of the cortex with respect to adrenal androgens and glucocorticoids, and indirect evidence about the hormones controlling electrolyte metabolism.

DR. H. M. WHYTE: I have a comment I should like to make in relation to what has come up in discussion. It concerns Dr. Evans's point regarding albuminuria, or lack of albuminuria, in the presence of oedema. Now, first of all, the urine was not tested, as far as we know, twelve months before this illness—that is, when she had cardiac failure. We do not know what the urine showed then. It was only on her admission to hospital that the urine was said to be free from albumin. I wonder how much faith we can put in this, because she must, at that time, have been hypoproteinaemic. In fact, her condition did not change very much from then until urine examinations repeatedly showed albumin. In other words, I wonder if the urine was really tested, or whether at the time of testing she was

experiencing a diuresis, which diluted the urine so that the protein concentration was reduced to such a degree that it was not distinguishable by the ordinary test. We have not infrequently found this in nephrosis. Another vague possibility is that there was Bence-Jones protein which might have been missed.

Pathological Report.

DR. E. HIRST: The patient was a thin woman of Polynesian extraction. There was well-marked pigmentation of the palmar creases. The exposed and the pressure areas showed no increased pigmentation, and an old abdominal scar was paler than the surrounding skin.

The relevant gross findings were as follows. The adrenal glands were normal in size, shape and weight. There was some fibrous thickening of the pleura of the right lung. There was no evidence of pulmonary tuberculosis, and the small calcified area in the left lung was unknown to us and not found. Situated on the right side of the middle mediastinum and projecting into the right pleural cavity there was a matted mass of caseating tracheo-bronchial and mediastinal lymph nodes, thought to be tuberculous.

The kidneys were small and unequal. There was some granularity of the subcapsular surface, and the architecture of the cut surface was blurred. The heart was enlarged. There was a small saccular aortic aneurysm. The mucosa of the colon was thickened and congested, and in the rectum submucosal thickening was palpable. The spleen was slightly enlarged, with a dry red cut surface.

Microscopic examination showed acid-fast bacilli in the caseous mediastinal lymph nodes.

Secondary generalized systemic amyloidosis was found in the following organs. The kidneys showed extensive deposits in almost all of the glomeruli and in relation to blood vessels. The spleen was notably involved by amyloid deposits in the red and white pulp. The palpable thickening in the rectum was due to amyloid deposit in the submucosa and *substantia propria*. Similar deposit was present in the remainder of the colon, and in the mucosa of the urinary bladder.

Amyloid deposit was found in the heart, replacing the cardiac muscle fibres in a manner similar to the replacement of the muscle of the vessel wall in other organs.

The adrenal and liver parenchyma was not involved. The vessels of the adrenal and the liver showed amyloid deposit in their walls, as did those of many other organs. In the parathyroids there was amyloid deposit causing parenchymal atrophy, and a microscopic eosinophilic adenoma was present.

Diagnosis.

Mediastinal lymph-node tuberculosis with secondary generalized amyloidosis, maximal in kidneys, spleen and colon.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

A CASE OF TETANUS.¹

[From the *Sydney Morning Herald*, October 13, 1870.]

A VERY SEVERE CASE of lockjaw has been successfully treated this week at the Parramatta Hospital. The patient had received a severe wound in the hand from a chaff-cutter 3 weeks ago. Several days after this symptoms of tetanus set in and the patient complained to his medical adviser. With great difficulty a small dose of croton oil and sugar was administered but little hopes were entertained of a cure. The doctor however continued to administer Calabar bean and brandy and during some days of unwearied attention was rejoiced to find the patient gradually recovering. Considering the severe nature of the case the cure deserves being recorded as a remarkable one.

¹ From the original in the Mitchell Library, Sydney.

Correspondence.

THE L.E. PHENOMENON IN RHEUMATOID ARTHRITIS.

SIR: Before a formidable list of clinical entities is classified into one group with the controversial L.E. phenomenon acclaimed as the common denominator, the laboratory technique for this test should be universally standardized and a rigid code of interpretation adhered to.

Lupus erythematosus is one example only of many conditions that sometimes have and sometimes have not the L.E. phenomenon as a laboratory associate. The term "L.E. phenomenon" therefore presents itself as the great misnomer and could well be renamed to indicate the actual significance of the test, if such is definitely known beyond all doubt.

More interesting is the attempt by the late L. J. A. Parr and others to provide a rationale for the use of antimalarial drugs in the treatment of rheumatoid arthritis. This pioneer investigation is still necessarily incomplete, and it will be most instructive when those still carrying on this study are able to provide the following data: (i) The total number of cases clinically acceptable as rheumatoid arthritis that were tested for L.E. phenomenon—not just the chosen 55. (ii) The total numbers of positive and of negative results. (iii) Of the positive results, the number that did not respond clinically to antimalarial drugs. (iv) Of the negative results, the number that did respond clinically to antimalarial drugs. (v) Of the positive cases treated with antimalarial drugs, in what percentage did the L.E. phenomenon disappear? (vi) What utilization was made in this series of other drugs and of important although often neglected physical means? Only with this information available can the value of the test as an indication for the antimalarial drugs be truly judged.

After six years of therapeutic trial of antimalarial drugs in rheumatoid arthritis, I have long been satisfied with their great value as one of a number of drugs with positive effect in this disease. No drug appears to have general application in rheumatoid disease; but if the presence of L.E. phenomenon is proved to be a reliable indication for treatment by mepacrine or "Chloroquine", it will signify an important accomplishment.

Yours, etc.,

149 Wickham Terrace,
Brisbane,
July 29, 1957.

JOHN A. SHANASY.

A PROTEST.

SIR: I feel it is time someone protested through your columns at the removal of *Mistura Calcii Acetylsalicylicis* (A.P.F.) from the list of pensioners' benefits. This high-handed bureaucratic action is apparently because the formula contains one minim of *Liquor Carmini*, which is not allowed.

The cost of one minim of *Liquor Carmini* is incalculably small, and this is bureaucratic hair-splitting with a vengeance. It means that we must write out the full formula, substituting *Tinctura Cardamomi Composita* for the *Liquor Carmini*—more waste of time.

Yours, etc.,

Wickham Street,
Ayr,
North Queensland.
July 24, 1957.

ROBERT NELSON.

THE DOCTOR-PATIENT RELATIONSHIP AND WORKERS' COMPENSATION.

SIR: The increasing and blatant infringement of human and professional dignity incorporated in the *Workers' Compensation Act, 1926-1955*, of New South Wales surely needs exposure to the clear light of medico-legal discussion.

Section 51 of the Act requires an injured worker to submit himself to examination by any registered medical person hired by an insurance company for the employer (presumably as a check on the integrity of the patient's own doctor).

This would be tolerable if the patient were seen in consultation with the attending doctor, but, as I am led to believe, consultation is rarely carried out in industrial practice.

It may be objected that consultations are difficult to arrange; but checking on one doctor by another should be so rarely necessary that the difficulty could be overcome. After all, the implication is that the patient's doctor may be issuing an improper certificate (which, being infamous conduct in a professional respect, could lead to his erasure from the medical register—surely itself a sufficient deterrent).

Yours, etc.,

607 New South Head Road,
Rose Bay,
New South Wales.
August 4, 1957.

GODFREY HARRIS.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course: Additional Lectures.

THE PostGraduate Committee in Medicine in the University of Sydney announces that the following additional lectures have been arranged for the annual subscription course:

Professor Robert Cruickshank, Director of the Wright-Fleming Institute of Microbiology, St. Mary's Hospital Medical School, London, an eminent epidemiologist and bacteriologist, will visit Sydney from August 20 to 24, 1957. By arrangement with the College of Pathologists of Australia, Professor Cruickshank will give the following lectures in the annual subscription course: Tuesday, August 20, 8.15 p.m., Maitland Lecture Theatre, Sydney Hospital: "The Epidemiology and Control of Rheumatic Fever." Wednesday, August 21, 2.15 p.m., seminar, Maitland Lecture Theatre, Sydney Hospital: "Hospital Infection." Friday, August 23, 1.15 p.m., Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital: "Recent Studies on Non-Specific Immunity."

Professor Graham Bull, of the Department of Medicine, Queen's University, Belfast, and Sir Edwin M. Tooth, Guest Professor for 1957 to the Brisbane General Hospital, will visit Sydney from August 21 to 24, and will give the following lecture on Thursday, August 22, at 8.15 p.m., in the Maitland Lecture Theatre, Sydney Hospital: "The Management of Renal Failure, with Special Reference to Acute Tubular Necrosis."

Professor R. J. Kellar, M.B.E., F.R.C.S., F.R.C.O.G., Professor of Obstetrics and Gynaecology, University of Edinburgh, will visit Sydney from August 24 to September 7. His programme will be as follows: Monday, August 26, 8.15 p.m., Stawell Hall, 145 Macquarie Street, Sydney: "Pregnancy Toxæmia." Wednesday, September 4, 8.15 p.m., Stawell Hall: "The Endocrine Control of the Menstrual Cycle in Health and Disease." Wednesday, September 4, 2 p.m., Royal Hospital for Women: "Carcinoma of the Cervix" (by arrangement with the New South Wales State Cancer Council, this lecture will be open to all members of the medical profession). Thursday, September 5, 1.30 p.m., King George V Memorial Hospital for Mothers and Babies: "Studies in Normal and Abnormal Micturition in the Female." Professor Kellar will also lecture at the Royal Newcastle Hospital on Sunday, September 1, at 8 p.m., on "Studies on Placental Function in Normal and Abnormal Pregnancy."

Colloquia on Hypertension and Pregnancy Toxæmia.

Colloquia on hypertension and pregnancy toxæmia will be held in Sydney during August, 1957, and the following sessions, to be held in the Stawell Hall, 145 Macquarie Street, Sydney, will be open to members of the subscription course: Monday, August 26: 8.15 p.m., "Pregnancy Toxæmia", Professor R. J. Kellar (Edinburgh). Wednesday, August 28: 9.30 a.m., "The Behaviour of Hypertensive Disease in Pregnancy", Professor Lance Townsend (Melbourne); 10 a.m., "The Prevention of Pregnancy Toxæmia", Dr. R. B. C. Stevenson (Sydney); 11 a.m., "The Treatment of Hypertensive Disease during Pregnancy with Proveratrine", Professor H. M. Carey (Postgraduate School of Obstetrics and Gynaecology, Auckland, New Zealand); 11.40 a.m., "The Aetiology of Preeclamptic Toxæmia and Eclampsia", Emeritus Professor F. J. Browne (Sydney); 2.15 p.m., open

forum (chairman, Dr. S. A. Smith; panel, Professor J. G. Hayden, Dr. G. B. Mackness, Dr. J. K. Maddox, Professor B. T. Mayes, Professor F. H. Smirk, Dr. R. B. C. Stevenson, Professor Lance Townsend).

Lectures by Professor C. A. Wells.

Professor C. A. Wells, Professor of Surgery, University of Liverpool, and McIlraith Guest Professor to the Royal Prince Alfred Hospital for 1957, will give the following lectures by arrangement with the Royal Prince Alfred Hospital and the trustees of the McIlraith Guest Professorship Fund: Monday, September 2, 8.15 p.m., Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital: "Duodenal Ulcer." Thursday, September 12, 8.15 p.m., Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital: "Hernia."

Lectures by Sir Russell Brock.

Sir Russell Brock, M.S., F.R.C.S., Surgeon to Guy's Hospital and Brompton Hospital, London, and Guest Surgeon to St. Vincent's Hospital, Sydney, is expected to visit Sydney from September 13 to October 6, 1957, and will give the following lectures: Thursday, September 19, 8 p.m., Stawell Hall, 145 Macquarie Street (in conjunction with the Laennec Society): "Bronchial Carcinoma." Wednesday, September 25, 8.20 p.m., Stawell Hall, 145 Macquarie Street (in conjunction with the Australian and New Zealand Cardiac Society): "The Basic Principles in Cardiac Surgery." Wednesday, October 2, 8 p.m., Stawell Hall, 145 Macquarie Street (under the auspices of the New South Wales State Committee of the Royal Australasian College of Surgeons): "Chest Injuries."

Post-Graduate Conference at Albury.

The Post-Graduate Committee in Medicine in the University of Sydney announces that, in conjunction with the Border Medical Association, a post-graduate conference will be held at the Albury Base Hospital on Saturday and Sunday, August 31 and September 1, 1957. The programme is as follows:

Saturday, August 31: 2 p.m., registration; 2.30 p.m., "Various Therapeutic Patterns", Dr. F. L. Ritchie; 4 p.m., "Treatment of Common Dermatoses", Dr. A. G. Finley.

Sunday, September 1: 9.30 a.m., "Modern Treatment of Anemias and Blood Dyscrasias", Dr. T. I. Robertson; 11 a.m., "The Management of Hypertension", Dr. F. L. Ritchie; 2.30 p.m., "Recent Trends in Dermatology", Dr. A. G. Finley; 4 p.m., "Treatment of Medical Complications in Pregnancy", Dr. T. I. Robertson.

The fee for attendance is £3 3s., and those wishing to attend are requested to notify Dr. R. Hayter, Honorary Secretary, Border Medical Association, North Street, Albury. Telephone: Albury 557.

FEDERATION OF COUNTRY LOCAL ASSOCIATIONS.

Sixth Post-Graduate Course, Maitland.

THE Federation of Country Local Associations (New South Wales) will hold its sixth post-graduate course at Maitland from September 16 to 20, 1957. The programme is as follows:

Monday, September 16: 9 a.m., registration; 10 a.m., official opening; 11.30 a.m., Memorial Oration ("The Late Dr. J. J. Hollywood and His Times"), Dr. F. P. M. Solling; 2.15 p.m., "The Management of Epilepsy in General Practice", Dr. K. B. Noad; 4 p.m., "Recurrent and Persistent Bronchitis", Dr. J. Beveridge.

Tuesday, September 17: 9.30 a.m., "The Modern Uses of Blood and Blood Products", Dr. R. J. Walsh; 11.30 a.m., "The Acute Abdomen", Dr. E. A. Hedberg; 2.15 p.m., symposium, "The Diagnosis and Treatment of Anæmia", Dr. R. J. Walsh and Dr. J. Beveridge; 4 p.m., "The Management of Pyelitis and Pyelonephritis and Their Complications", Professor C. R. B. Blackburn; evening, open lecture, Sir Ernest Fisk.

Wednesday, September 18: 9.30 a.m., "Surgical Emergencies in General Practice", Dr. K. C. T. Rawle; 11.30 a.m., "Treatment of Heart Failure in Practice", Dr. J. G. Radford.

Thursday, September 19: 9.30 a.m., "The Anatomy and Surgery of Inguinal Hernia", Dr. E. A. Hedberg; 11.30 a.m., "Treatment of Fractures of the Lower Extremity", Dr. H. Barry; 2.15 p.m., symposium, "The Painful Back", Dr. K. B.

Noad, Dr. E. A. Hedberg, Dr. H. Barry; 4 p.m., "The Ischæmic Limb", Professor John Loewenthal.

Friday, September 20: 9.30 a.m., "Migraine and Allied Neuro-Vascular Disturbances", Dr. K. B. Noad; 11.30 a.m., "Drugs in Obstetric Practice", Dr. R. J. Gill; 2.15 p.m., "Emergencies in the Newborn Period", Dr. John Beveridge; 4 p.m., meeting of delegates, medico-political conference.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. have been promulgated in the *Commonwealth of Australia Gazette*, No. 38, of July 4, 1957.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

To be Surgeon Commander.—Surgeon Lieutenant-Commander John Arthur Basil Cotsell (Acting Surgeon Commander).

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Resignation.—The resignation of Eugene Peter O'Sullivan of his appointment as Surgeon Lieutenant is accepted, dated 6th May, 1957.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—1/33136 Captain (provisionally) K. F. Brady relinquishes the provisional rank of Captain, 12th March, 1957, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command), in the honorary rank of Captain, 13th March, 1957. To be Temporary Lieutenant-Colonel, 28th May, 1957: 1/39206 Major W. D. Exton. To be Captain (provisionally), 17th May, 1957: 1/46932 Noel Clarkson Holmes.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/147974 Lieutenant-Colonel C. R. B. Blackburn is appointed from the Reserve of Officers, is appointed Consultant Physician, Army Headquarters, and to be Colonel, 12th April, 1957. The provisional rank of 2/127049 Captain G. R. W. McDonald is confirmed. To be Temporary Major, 14th May, 1957: 2/127807 Captain J. B. Westphalen.

Southern Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 3/101834 Captain B. W. Fox is confirmed. To be Major, 30th April, 1957: 6/15245 Captain B. A. Smithurst.

Central Command.

Royal Australian Army Medical Corps (Medical).—The regimental seniority of 4/31952 Major R. A. Kenihan is in accordance with Army seniority (24th June, 1955).

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—6/5128 Major D. B. Nathan is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Tasmania Command), 17th May, 1957.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Northern Command.—To be Honorary Captains: John Howard Casey, 17th May, 1957, Colin James Claxton, 20th May, 1957, and Hugh Olivey Beeston, 24th May, 1957.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Wing Commander L. R. Trudinger (033059) is promoted to the rank of Group Captain.

The following Flight Lieutenants are promoted to the rank of Squadron Leader: M. A. May (013705) (Acting Squadron Leader), N. L. Groves (025211).

The following appointments, changes etc. have been promulgated in the *Commonwealth of Australia Gazette*, No. 41, of July 18, 1957.

AUSTRALIAN MILITARY FORCES.

Citizen Military Forces.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/101028 Captain (provisionally) D. M. O'Sullivan is seconded whilst in the United States of America, 15th February, 1957. The provisional rank of 3/87695 Captain J. G. Bunday is confirmed. 3/101819 Captain (provisionally) J. H. S. Martin relinquishes the provisional rank of Captain, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Southern Command), and is granted the honorary rank of Captain, 6th March, 1957. The provisional appointments of the following officers are terminated: Captains 3/50197 W. A. Syme, 26th July, 1956, 5/26400 M. N. Orton, 14th November, 1956, 3/101031 R. C. W. Williams, 9th January, 1957, and 3/101028 D. M. O'Sullivan and 3/101029 J. M. Court, 14th February, 1957. To be Captains (provisionally): 3/50197 William Archibald Syme, 27th July, 1956, 5/26400 Mervyn Noel Orton, 15th November, 1956, 3/101031 Robin Charles Winfield Williams, 10th January, 1957, and 3/101028 David More O'Sullivan and 3/101029 John Maurice Court, 15th February, 1957.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps (Medical).

Southern Command.—The age for retirement of Major D. E. Gowenlock is extended until 20th June, 1958.

The following officers are placed upon the Retired List, with permission to retain their rank and wear the prescribed uniform:

Southern Command.—Major C. B. Berryman and Captains I. D. McInnes and F. R. Vincent, 27th May, 1957, and Lieutenant-Colonel (Honorary Colonel) E. T. T. Downie, Major W. E. A. Hughes-Jones, and Captains J. R. Heath and G. R. A. Syme, 1st July, 1957.

Central Command.—Major G. M. Hone, 31st May, 1957, and Colonel (Honorary Brigadier) D. M. Salter, E.D., Lieutenant-Colonel M. T. Cockburn, Majors I. A. Hamilton, W. R. C. Morris and N. T. M. Wigg, and Captains M. E. Chinner, C. M. Deland, A. T. Harbison, A. D. Reid and J. B. Wilson, 30th June, 1957.

The following officers are retired:

Southern Command.—Honorary Captains S. M. Hall, D. B. Rosenthal and M. E. Thornton, 27th May, 1957, and Honorary Captain J. C. Spencer, 1st July, 1957.

Central Command.—Honorary Captains R. D. Hornabrook and R. E. Drever, 31st May, 1957, and Honorary Captains A. D. Cocks, A. W. Grote, H. R. R. Hancock and G. Tassie, 30th June, 1957.

Western Command.—Honorary Captains M. S. Bell and P. W. Shanahan, 31st May, 1957.

The following appointments have been promulgated in the *Commonwealth of Australia Gazette*, No. 42, of July 25, 1957.

ROYAL AUSTRALIAN AIR FORCE.

His Excellency the Governor-General has been pleased to approve the appointment of the undermentioned officer in the capacity shown:

To be Honorary Surgeon to the Governor-General: Group Captain Lawrence Robert Trudinger, M.B., B.S., D.T.M. & H., vice Group Captain George Charles Victor Thompson, M.B., B.S., F.R.C.S. (Ed.).

Her Majesty the Queen has been graciously pleased, under date 1st July, 1957, to approve the appointment of the undermentioned officer in the capacity shown:

To be Honorary Surgeon to the Queen: Group Captain George Charles Victor Thompson, M.B., B.S., F.R.C.S. (Ed.), vice Group Captain Clifford Henry Coomer Searby, B.Sc., M.B., M.S., F.R.C.S. (Eng.), F.R.A.C.S.

The following appointments, changes etc. have been promulgated in the *Commonwealth of Australia Gazette*, No. 43, of August 1, 1957.

ROYAL AUSTRALIAN AIR FORCE.

Air Force Reserve.

Medical Branch.

The following former officer is appointed to a commission, 13th May, 1956, with the rank of Flight Lieutenant: K. F. Brennan (253473).

The following are appointed to a commission with the rank of Flight Lieutenant: George Jack Burgess (018411), 29th January, 1957; Peter Henry Caldwell (257972), 2nd May, 1957.

The provisional appointment of the following Pilot Officers is confirmed and they are promoted to the rank of Flight Lieutenant: W. A. C. Douglas (015183), 10th December, 1956; G. W. Harley (034792), J. P. McCarthy (0311615), 18th December, 1956; G. G. Farrant (034796), 19th December, 1956.

Flight Lieutenant (Temporary Squadron Leader) W. D. Cunningham (261896) is promoted to the temporary rank of Wing Commander, 2nd May, 1957.

Flight Lieutenant I. C. Morrison (0210985) is granted the acting rank of Squadron Leader, 30th October, 1956.

The following Flight Lieutenants (Temporary Wing Commanders) relinquish the temporary rank of Wing Commander and are placed on the Retired List, 25th October, 1956: N. M. Cuthbert, M.C. (1465), J. O'Sullivan (2076), B. W. B. Riley (264370), A. J. H. Stobo (2100).

Flight Lieutenant (Temporary Squadron Leader) A. B. K. Watkins (2060) relinquishes the temporary rank of Squadron Leader and is placed on the Retired List, 25th October, 1956.

The appointment of the following officers is terminated: Flight Lieutenant (Temporary Squadron Leader) C. Craig (1849), 25th October, 1956; Flight Lieutenants R. E. B. Brown (4854), 25th October, 1956, and A. I. Lane (0210562), 12th November, 1956.

University Intelligence.

UNIVERSITY OF MELBOURNE.

Elections to Council and Standing Committee of Convocation by Graduates.

Notice is hereby given that nominations will be received up to 4 p.m. on Monday, September 30, 1957, to fill five vacancies for representatives of graduates on the Council of the University of Melbourne and 20 vacancies for representatives of graduates of all faculties on the Standing Committee of Convocation. Nomination forms may be obtained from the Returning Officer.

UNIVERSITY OF ADELAIDE.

The Shorney Prize.

THE SHORNEY PRIZE, established for the purpose of perpetuating the memory of the late Herbert Frank Shorney, M.D., F.R.C.S., Lecturer in Ophthalmology in the University of Adelaide from 1926 to 1933, will be offered for the ninth time in 1958, and will be for work in diseases of the ear, nose and throat.

The relevant clauses of the statute are as follows:

3. A post-graduate prize, to be called The Shorney Prize, of the value of £100, shall be awarded to the candidate who in the opinion of the examiners has made the most substantial contribution to knowledge in the subjects of ophthalmology or of diseases of the ear, nose and throat.

4. The recipient must be a graduate of an Australian university.

5. The material submitted for the prize may be either a thesis or published work in medical or scientific literature.

6. Each candidate must declare that the work described is his own.

7. The prize shall be offered for competition from time to time as the accumulations of the fund permit.

8. The prize shall be offered at least twelve months before the last day for the receipt of applications.

9. The prize shall not be awarded on any occasion unless in the opinion of the examiners the material submitted is of sufficient merit.

Applications, accompanied by three copies of the evidence which the candidates wish to submit in support, must reach the Registrar, University of Adelaide, not later than July 31, 1958.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following additions and amendments have been made to the Register of Medical Practitioners for New South Wales, in accordance with the provisions of the *Medical Practitioners Act, 1938-1957*:

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (1) (a) of the Act: Bradshaw, Alan Robert, M.B., B.S., 1951 (Univ. Queensland); Tsiang, Ching-Tang, M.B., B.S., 1955 (Univ. Melbourne).

Registered medical practitioner who has complied with the requirements of Section 17 (3) and is registered under Section 17 (1) (b) of the Act: Hingston, Richard George, M.B., B.Ch., 1956 (National Univ., Ireland).

Registered medical practitioners who have complied with the requirements of Section 17 (3) and are registered under Section 17 (2) (a) of the Act: Berkowicz, Leon, M.D., 1947 (Univ. Milan); Kraus, Otto, M.D., 1919 (Univ. Prague).

The following additional qualification has been registered: Kelly, Kenneth Melville, M.F.A., R.A.C.S., 1952.

Registered medical practitioners who are required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and are registered under Section 17 (1) (a) of the Act: Mathias, Nansi Avis, M.B., B.S., 1956 (Univ. Sydney); Froggatt, Eric James, M.B., B.S., 1957 (Univ. Sydney); George, Rena, M.B., B.S., 1957 (Univ. Sydney); Jackson, Geoffrey William Theodor, M.B., B.S., 1957 (Univ. Sydney); Koller, Karl Max, M.B., B.S., 1957 (Univ. Sydney); Lye, Tong Meng, M.B., B.S., 1957 (Univ. Sydney); Phillips, John Keith Andrew, M.B., B.S., 1957 (Univ. Sydney); Thompson, Graham Stuart, M.B., B.S., 1957 (Univ. Sydney); Watson, William Joseph, M.B., B.S., 1957 (Univ. Sydney); Young, Gavan Bernard, M.B., B.S., 1957 (Univ. Sydney); Young, Ian Frederick, M.B., B.S., 1957 (Univ. Sydney).

Registered medical practitioner who is required to complete twelve months' hospital service in accordance with the provisions of Section 17 (3) and is registered under Section 17 (1) (c) of the Act: Pavlovic, Leopold, M.D., 1948 (Univ. Prague).

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

THE General Secretary of the Australasian Medical Congress (British Medical Association), Tenth Session, has sent the following notes for publication.

Exhibition of Doctors' Hobbies.

At the forthcoming Congress to be held in Hobart from March 1 to 7, 1958, there will be an exhibition of doctors' hobbies, and the organizers are most anxious to have exhibits from members in all States. Intending exhibitors may be assured that every care will be taken of exhibits, and arrangements will be made for exhibits to be covered by insurance.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 27, 1957.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	2	1(1)	1	4
Amoebiasis
Ancylostomiasis	4	4
Anthrax
Bilharzias
Brucellosis	1	1
Cholera	1	1
Chorea (St. Vitus)	1	1
Dengue
Diarrhoea (Infantile)	2(1)	14(10)	16
Diphtheria	1(1)	3(1)	5
Dysentery (Bacillary)	5(2)	5
Encephalitis	1(1)	1(1)	2
Filariasis
Homologous Serum Jaundice	1
Hydatid	1(1)	1
Infective Hepatitis	41(12)	44(22)	1(1)	6(5)	8(1)	2(1)	1	..	103
Lead Poisoning	1	2
Leprosy	1	..	1
Leptospirosis	4	4
Malaria
Meningococcal Infection	3(2)	3(1)	1	7
Ophthalmia
Ornithosis
Paratyphoid
Plague
Poliomyelitis
Puerperal Fever	1	1
Rubella	66(44)	..	14(8)	2(2)	..	1	..	83
Salmonella Infection
Scarlet Fever	13(7)	13(14)	11(2)	11(7)	2(2)	55
Smallpox
Tetanus	2	..	4	..	6
Trachoma
Trichinosis
Tuberculosis	39(24)	22(18)	7(3)	2(1)	6(3)	6	82
Typhoid Fever
Typhus (Flea-, Mite- and Tick-borne)
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

Interested members are asked to get into touch as soon as possible with the chairman of the Hobbies Exhibition, Dr. R. J. Hudson, 71 Main Road, Moonah, Hobart, Tasmania.

Public Health.

A VIRULENT STAPHYLOCOCCAL INFECTION ACCOMPANYING INFLUENZA.

THERE has been in the last few weeks in Sydney an extremely virulent infection caused by the *Staphylococcus aureus*. Apparently similar infections have occurred in other States as well. As this particular type of infection has been present only for the same time as the prevailing influenza epidemic, it would appear that there is an association between the two.

It is possible that staphylococcal pneumonia, with or without septicaemia, occurs within the first few hours of the influenzal infection, the patient up to this time having been a carrier only of the staphylococcus, probably in the nose.

The syndrome is characterized by rapid onset of fever, toxæmia, circulatory collapse with pallor and cyanosis, hæmoptysis, and occasional respiratory obstruction in the trachea and major bronchi. Profound neutropenia may be a feature. Death may occur within twenty-four hours, particularly in patients under the age of twenty-five years. The clinical picture is sufficiently striking for a diagnosis to be made without waiting for bacteriological confirmation.

Treatment is a matter of real urgency, and intravenous administration of broad-spectrum antibiotics is indicated. The preferred antibiotics are chloramphenicol, erythromycin and tetracycline. Immediate admission to hospital is desirable.

Notice.

SPECIAL GROUP ON AVIATION MEDICINE (BRITISH MEDICAL ASSOCIATION).

A SCIENTIFIC MEETING of the Special Group on Aviation Medicine (British Medical Association) is to be held on Thursday, September 5, 1957, at 8 p.m., in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney.

The meeting will include an address by Squadron Leader James McC. Morrison, Royal Australian Air Force, entitled "The Doctor and the Flier in Canada and the United States". Squadron Leader Morrison is attached to the staff of the School of Aviation Medicine, Point Cook, and his address will cover his observations and experiences while on a recent tour of duty in the United States of America.

All medical practitioners are cordially invited to be present.

Medical Appointments.

Dr. J. H. W. Birrell has been appointed Assistant Government Pathologist at the City Coroner's Court, Melbourne.

Dr. E. F. West has been appointed a member of the Physiotherapists Board of South Australia.

Dr. H. K. Fry has been appointed an official visitor at the Parkside Mental Hospital, South Australia.

Dr. L. R. H. Drew and Dr. E. F. Fitzpatrick have been appointed medical officers in the Mental Hygiene Branch Department of Health, Victoria.

Dr. G. R. Weigall has been appointed a trustee of the land permanently reserved on March 14, 1882, as a site for a hall and library for the use of the Medical Society of Victoria and for other scientific purposes, at East Melbourne, Victoria.

Dr. H. A. Handley has been appointed Honorary Clinical Assistant, Ophthalmological Department, Royal Adelaide Hospital.

Dr. A. A. Tye has been appointed Honorary Ophthalmologist in the Parkside Mental Hospital, South Australia.

Dr. G. R. Whittle has been appointed Government Medical Officer at Kingaroy, Queensland.

Dr. R. Schaefer has been appointed officer of health to the local board of health of East Murray, South Australia.

Dr. S. G. Sandas has been appointed Medical Superintendent in the Division of Mental Hygiene, Department of Public Health, New South Wales.

The following have been appointed to be Medical Officers, Mental Hygiene Branch, Department of Health, Victoria: Dr. O. H. D. Blomfield, Dr. K. H. Bryant, Dr. H. G. Edhouse, Dr. N. K. Honey, Dr. C. Elliot.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Dorman, George Alan, M.B., B.S., 1955 (Univ. Sydney), 25 Crescent Street, Haberfield, New South Wales.

Morgan, Brian Patrick, M.B., B.S., 1953 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown, New South Wales.

Diary for the Month.

- Aug. 19.—Victorian Branch, B.M.A.: Finance Subcommittee.
- Aug. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- Aug. 21.—Victorian Branch, B.M.A.: Clinical Meeting.
- Aug. 21.—Western Australian Branch, B.M.A.: General Meeting.
- Aug. 22.—New South Wales Branch, B.M.A.: Clinical Meeting.
- Aug. 22.—Victorian Branch, B.M.A.: Executive of Branch Council.
- Aug. 23.—Queensland Branch, B.M.A.: Council Meeting.
- Aug. 27.—New South Wales Branch, B.M.A.: Ethics Committee.
- Aug. 28.—Victorian Branch, B.M.A.: Branch Council.
- Aug. 29.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, 88 L'Estrange Terrace, Kelvin Grove, Brisbane, W.1.): All applicants for Queensland State Government Insurance Office positions are advised to communicate with the Honorary Secretary.

South Australian Branch (Honorary Secretary, 86 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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